THE BOSTON
Medical and Surgical
JOURNAL

VOLUME 192 FEBRUARY 26, 1925 NUMBER 9

ORIGINAL ARTICLES

THE OPERATIVE TREATMENT OF VARICOSE ULCER*

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INTRODUCTION

VARICOSE ulcers are to be expected in those suffering from varicose veins who, for one reason or another, are unable to protect themselves against injury and infection. The slowness in healing so noticeable in wounds and abrasions of the legs even of normal individuals is greatly intensified by the passive congestion due to varicosity of the superficial veins. I use the word "superficial" advisedly since the deep veins so rarely become varicose that they may be counted on not only to carry on their own functions but such labors as are abandoned by the veins external to the deep fascia.

In earlier writings† I have discussed the physiology of the venous circulation in the legs but it may not be out of place here to speak of the changes brought about by varicosity of the great saphenous vein and its tributaries and the relation of varix to ulcer. Normally, blood is carried away from the legs by (1) suction due to respiration and the heart beat, by (2) the arterial push through the capillary system and (3) most important of all, by the action of the muscles of the legs upon the veins themselves. It is of course owing to the presence of numerous bicuspid valves within the veins that muscular pressure upon them is able to force the blood onward toward the heart, and it is to the destruction of those valves by excessive back pressure that varix is due. Moreover it is clear that the veins within the deep fascia, surrounded as they are by muscles, will not nearly so easily become disabled by back pressure as is the case with the superficial veins.

Once the surface vessels lose the use of their valves they become reservoirs for venous blood which pours down them more readily than it mounts through them, and indeed it is not improbable that a local reversal of the circulation occurs in the great saphenous system whereby blood spills over from the femoral vein into the great saphenous at the groin, passes down toward the foot, reënters the deep system, passes up again to the groin and so on. In any case it is clear that all superficial tissues drained by a varicose vein are subject to marked passive congestion so long as the subject is sitting or standing, and that products of tissue metabolism are inefficiently removed.

That the skin does not more often show signs of malnutrition in the presence of varix is due primarily to the excellence of the deep circulation and to the presence of a number of vessels connecting the superficial with the deep veins. These are spoken of as perforating veins. Their valves are so set as to allow a flow of blood only from without inward and accordingly they act as safety valves for the dilated surface veins. They are very variable in number and are found for the most part in the lower leg. The lesser saphenous vein which empties into the popliteal vein in the popliteal space may be regarded as one of them. Occasionally these veins become varicose, particularly as a result of superficial phlebitis, and in that case ulceration is almost certain to occur.

For purposes of treatment, an elaborate classification of ulcers is quite unnecessary. They may take a great variety of forms; round and superficial or irregular and deep, small or large, clean or dirty, depending upon a variety of circumstances. But in relation to their underlying cause—varicose veins—they fall into two classes, distinct in many ways. In the first class are the ulcers dependent upon the common superficial varicose vein. In the second are the ulcers secondary to thrombo-phlebitis. The ulcers of the first class are usually found riding upon or at the foot of a large varicose vessel. The ulcers of the second, not infrequently multiple, are seldom obviously related to visible veins and may appear in unusual positions. The appearance of the ulcers themselves may be precisely alike. It is the veins which are different.

ULCERS ASSOCIATED WITH SIMPLE VARIX

The history of these ulcers is very familiar to everyone. As a rule the patient has suffered for years from varicose veins. There may have been present one great tortuous vessel extending from groin to ankle, or the front and inner side of the calf may be covered with groups of varicose veins. There may have appeared,

*Read before the Brockton Medical Society, November 13, 1924.

†Surgery, Gynecology and Obstetrics, Vol. xxii, p. 143, 1916.

from time to time, pigmented patches over some of these veins indicating chronic irritation of the skin. The actual beginning of the ulcer is usually to be found in a blow, an abrasion from scratching or even an actual rupture of a vein. The ulcer is at first small, shallow and readily healed by bandaging or rest in bed. If neglected, it may become large, deep and surrounded by a zone of induration and oedema. But always there will be felt beneath it, or on the proximal side of it, a distended vein, sometimes a great "lake" of blood.

The level at which these ulcers usually become established is the middle third of the lower leg. They never appear near the knee and only occasionally upon one side or the other of the ankle. It seems as if the ulcer tended to occur where the aggregate capacity of the veins was greatest, before they had become gathered into a few vessels, that is, at a point where the circulation was the slowest. Naturally, the front and inside of the calf is the point most eagerly scratched and most frequently bruised, but injury alone does not explain the frequency of ulcer in the middle third of the leg.

In deciding upon a method of treating one of these ulcers, the surgeon may be sure that thorough removal of the varicose veins will, if properly performed, always do good and never, barring the rare fatal accidents of surgery, do harm. On most occasions, removal of the varicose veins from the saphenous opening to a point just above the ulcer will effect a cure. Under some circumstances excision of the ulcer as well is required. Examinations made before operation will settle this point.

Method of Examination and Operative Indications—The size of the ulcer is not particularly important but the amount of indurated tissue about and beneath it is of great significance, for if scar tissue about an ulcer is very deep and extensive it is unreasonable to expect prompt and permanent healing even if passive congestion is relieved by removal of the tributary vein or veins. Thus excessive induration is an indication for excision and skin graft.

Another almost more important factor is the presence of a varicose perforating vein in the region of the ulcer. For should one of these vessels remain after operation connected with a superficial varicose vein beneath the ulcer, local venous stasis will still be present even after removal of the varicose vessels above the level of the ulcer. Examination should therefore be made to detect the presence of one or more of these diseased perforating veins. This examination is relatively simple but requires some practise if it is to be interpreted with confidence.

After going over the patient carefully, inspecting the legs, noticing the size of the veins and their relation to the ulcer and deciding upon the amount of induration of the ulcer itself, make yourself familiar by both sight and touch with the amount of tension in the distended veins when the patient is standing. Then have the patient sit down in a chair which has a strong back, a rocking chair preferred. By tipping back the chair and raising the leg, the veins are emptied of blood. As a rule, they are now quite palpable but utterly relaxed. The chair is then tipped forward, the leg dropped to the floor and the patient stands up. Varicose veins fill almost with a shock since the blood pours rapidly into them from above. This test, devised by Trendelenburg, confirms the diagnosis of varicose veins but gives no information as to the presence of leaky perforating vessels. Accordingly it is repeated in a somewhat different way. After the leg has been emptied of blood by elevation, a piece of gauze bandage is passed about the upper thigh and its ends brought out between the fingers in the same way that reins are held in the hand in driving a horse. A half twist of the hand is sufficient to compress the superficial veins. As soon as this compression is made the chair is tipped forward and the patient stands. There can now be no flow of blood down the varicose veins which, at first, remain collapsed but should there be any leaking of blood through the perforators from the deep veins to the surface, the varicose vessels will be seen and felt to fill quite rapidly, perhaps in ten, fifteen or twenty seconds. On the other hand, if the veins below the constriction remain flat and soft from one-half to one minute there can be no leaking perforator of any consequence and only gradually will the great veins fill from the capillary circulation. Even then there will be seen and felt upon the final release of the constriction a distinct additional swelling of the varicose veins below it. The French have called this test by constriction the counter-proof (contre-épreuve) of the Trendelenburg test.*

Operative Treatment—The application of this test to varicose ulcer is quite simple. Should the test disclose the presence of outward-leaking perforating vessels it is possible that one of these is in the neighborhood of the ulcer. This is an indication for excision of the ulcer together with the veins beneath it, and if the area left is too large to close without undue tension, a skin graft is required. I hardly need say that the excision of the veins above the level of the ulcer should be carried out and finished before the region of the ulcer is dissected, since the ulcer itself, and the tissues about it, are necessarily infected. Usually the Thiersch graft can immediately be applied, though it is optional, I think, to wait some ten days and graft upon fresh granulations. It is absolutely necessary, however, to excise the deep fascia behind the ulcer, whether muscle, tendon-sheath or bone is exposed. Otherwise the graft is likely to fail or to take poorly.

*This test is described and illustrated in the Boston Medical and Surgical Journal, Vol. clxxxvii, p. 258, 1922 (August 17).

The excision of varicose veins, whether or not in the presence of ulcer, should be done carefully and with gentleness though the exact method of so doing seems to me unimportant. The great saphenous vein should be tied and cut about half an inch below its entrance into the femoral and *all branches entering the stump carefully divided.* It may then be stripped or everted to a point below the knee. Below this point open dissection is to be preferred. Flaps should be turned up for a short distance and, as far as possible, the principal vein and its branches dissected from the under surface of the fat. In this way the danger of causing sloughs of the skin is done away with provided the flaps are not turned back more than an inch or so. Few surgeons are aware, I believe, that the small arteries which nourish the skin of the calf pass directly out from the vessels beneath the deep fascia, perforating it at quite regular intervals. They do not run up and down or across for any great distance. Thus to some degree they are terminal and do not permit the making of very extensive flaps. Should sloughing of the skin margins follow operation it indicates rough handling, infection or carelessly made flaps, any or all.

The treatment of the foregoing type of ulcer may be summed up very quickly. They are cured as a rule by thorough excision of the varicose veins. If they are very old and indurated, excision of the ulcer must be added to excision of the veins. Varicose perforating vessels will seldom be found in association with these ulcers but should always be sought for by the constriction test. If the test suggests their presence the ulcer should be excised. The deep fascia behind an ulcer should always be removed with it.

VARICOSE ULCERS RESULTING FROM PHLEBITIS

These ulcers are not common and indeed are unlikely to be recognized as being related to disease of the veins unless a careful history is taken. I believe that they are often regarded as syphilitic though they do not resemble any syphilitic ulcer with which I am familiar. They result from phlebitis which has occurred in veins not previously varicose. In women, "milk leg" following childbirth is the usual cause. In men, the phlebitis of typhoid fever or of any debilitating infection. As a rule the ulcers appear within a year following the phlebitis. Rarely, ulceration is delayed for so many years that the patient no longer associates the two diseases.

If you should see an individual suffering from one of these ulcers you would be struck by the fact that very few enlarged veins were visible. You would notice that the region of the ulcer was very much indurated but that the induration was not confined to this situation—rather that it was widespread in the lower leg, perhaps most noticeable upon the back of the calf or as a ring encircling the calf. And if you examined very carefully the position of the great saphenous vein between the knee and the groin, you might feel a small hard straight cord representing the healed phlebitic vessel.

In any instance of ulcer of the leg not apparently associated with varicose veins, inquire for a history of phlebitis in the past. Apparently the sort of phlebitis which occurs in veins not previously varicose* affects not only the veins themselves but results in a variable amount of thickening of the deep fascia of the calf and of the fat between it and the skin. Sometimes the fascia is so extensively diseased as to resemble the condition found in elephantiasis and I am inclined to believe that the ensuing induration and oedema are associated with, if not caused by, damage to the lymphatics secondarily involved in the extensive infection of the whole great saphenous system. In any

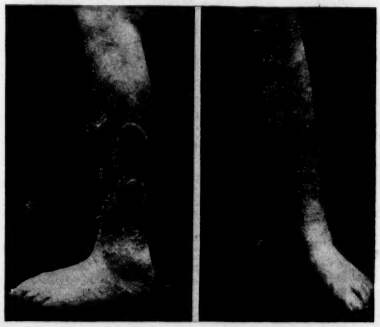

FIG. 1. M. C. C. Surg. No. 3597, Peter Bent Brigham Hospital. Post-phlebitic ulcer. Anterior and lateral views of the left leg. Notice absence of visible veins in both views, the areas of pigmentation on both internal and external surfaces of the calf and the unusual situation of the ulcers. Induration extended almost completely about the lower leg. The deep fascia was found at operation to be greatly thickened.

case, oedema and induration of the calf, together with a variable amount of swelling of the foot, are characteristic of the condition. Ulcers crop out in unexpected places—on the back of the calf or on the outer side of the ankle, for instance, and multiple ulceration is to be expected. Repeated attacks of cellulitis or lymphangitis are not uncommon. (Figures 1 and 2.)

A vein originally the seat of phlebitis is subsequently canalized. Thrombosis never occludes a vein for very long, but in the process of repair the valves are destroyed, the lumen narrowed and the wall thickened. In contrast with the dilated tortuous varicose vein of the common type, these vessels are small, straight and hard. They are incapable of transmitting

*Phlebitis in veins already varicose appears to remain confined to a few large principal vessels. Its remote effects are decidedly less harmful than those that result from phlebitis in veins previously normal, particularly if it is treated by excision of the veins in the subsiding stage of the disease.

blood in the right direction by the ordinary mechanism since they have no valves, and must be classed in this sense as varicose veins. There is no mistaking the character of these vessels when they are encountered in a dissection.

When such a condition is present it is hard to believe that the deep veins as well are not diseased. However, cyanosis in the foot has never, in my experience, been so marked as to suggest that the deep circulation is not doing

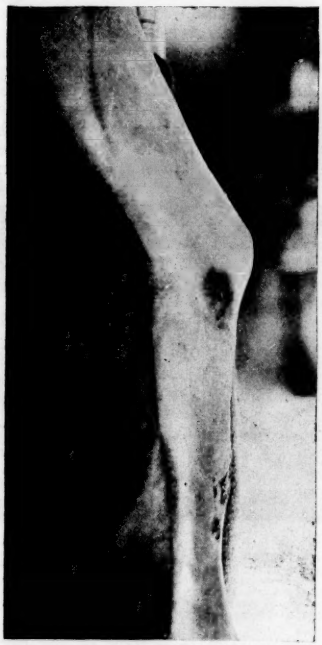

FIG. 2. J. T. M. Surg. No. 21729, Peter Bent Brigham Hospital. Post-phlebitic ulcer. View of internal surface of the left leg—the patient being in bed. Notice streak of lymphangitis in upper thigh. The actual ulcer at this time was comparatively small but the area of brawny induration and oedema was very large. A healed ulcer is seen just below the knee. The patient had been incapacitated for many months. Compare with Fig. 3 for result of treatment.

its work about as usual, and the removal by operation of the great saphenous vein certainly leaves the venous circulation improved rather than impaired.

The perforating veins are in many instances made useless by phlebitis. Instead of carrying blood inward they allow it to leak from the deep into the superficial vessels. This can usually be demonstrated by the constriction test provided a superficial vein can be found large and soft enough to examine. As a rule the superficial veins fill below the constriction, when the patient stands, in a few seconds.

It will be clear to you then that the ulcers which occur under these conditions are serious affairs. The superficial tissues of the calf, and especially the deep fascia, are more or less widely indurated. There is apt to be a greater extent of brawny oedema than would be expected to occur solely as a result of infection residing in and about such ulcers as are present. In tissue of this character, ulcers are likely to be widespread. Heal an ulcer in one place and another will soon appear elsewhere. The lower leg is sometimes encircled. But there is nothing characteristic about the appearance of the ulcer per se. Some of the most extensive are very shallow. Others are deep and surrounded by a sharply elevated oedematous wall. The skin about some is atrophied as a result of repeated attacks of cellulitis. About others it is thick and oedematous.

Treatment by bandaging is singularly ineffective and the reason for this is simple enough. Bandaging in the presence of varicose ulcer of the ordinary type compresses the dilated veins, forces the venous blood into smaller and more normal channels and relieves oedema. Thus circulation in and about an ulcer is improved. But in the case of post-phlebitic ulcer there are no dilated veins to compress and the induration of the calf is such that bandaging can in any case have very little effect. Local applications of an antiseptic sort may, however, help to some extent.

Operative Treatment requires careful preparation and planning, perfect technique and, what is almost more important than all these, the intelligent coöperation of the patient. It is wise to inform such patients that they may require more than one operation and that they may have to spend many weeks in hospital. That their ulcers should be permanently cured sometimes seems impossible, but that they will be improved, made comfortable and perhaps cured may safely be stated.

You will remember that the ulcer which rides upon a varicose vein is usually cured by excision of the varix, but must, on some occasions, be excised. Post-phlebitic ulcers are never cured by removal of the veins and must always be excised. Even then, more may be required. If, as appears to be the fact, a widespread phlebitis leaves the subcutaneous tissues indurated and the deep fascia thickened, scarred and shrunken upon the muscle beneath, it is probably true, as has already been suggested, that a condition has become established such as is seen in elephantiasis, the difference between the two states being one of degree and of a different manner of origin. For in elephantiasis the lymphatics are primarily diseased and thickening of the deep fascia, subcutaneous fat and skin is altogether dependent upon the choking of these vessels and the infections which follow. Post-phlebitic induration and oedema, on the other hand, result from a spreading of

infection to the lymphatics secondary to thrombo-phlebitis. The resulting conditions in the two diseases are not widely different and it occurred to me some time ago that since excision of great strips of deep fascia (Kondoleon operation) had proved to be the most effective means of treating elephantiasis, excision of deep fascia might prove of real advantage in the treatment of post-phlebitic disease. The results in two instances treated upon this principle have been encouraging, though I should not care as yet to be very positive upon this point.

FIG. 3. J. T. M. Surg. No. 21729, Peter Bent Brigham Hospital. Post-phlebitic varix. The ulcer area, together with the deep fascia, has been widely excised. Note the extent of the skin graft which half encircles the leg. The patient is back at work. Compare with Fig. 2, the same patient. The scars left after excision of the veins are almost invisible.

With these thoughts in mind, treatment of post-phlebitic ulcer may be outlined somewhat in the following way:

The patient is kept in bed until the maximum improvement in the inflammatory reaction appears to have been obtained. It is not necessary that the ulcer should have healed during this period which may extend over one to three weeks. The great saphenous vein is then removed down to the upper limit of the ulcerated area. Should the dissection of the upper calf be carried into indurated tissue, great care must be used in handling and closing the incision. This part of the operation is to be completed before anything further is done. The area of ulceration, including any thin, bluish, recently healed skin about it, may now profitably be excised taking with it in one mass the veins and deep fascia behind. This excision disregards altogether the possibility of exposing bone, tendon-sheath and muscle. It is surprising how unchanged the tissues behind the deep fascia appear, and it is undoubtedly important that the loose tissue immediately beneath the deep fascia should be preserved. All bleeding points are tied with the finest catgut or stopped by compression with hot moist gauze. The exposed area may then be covered with a Thiersch graft (Figure 3). This graft is likely to be successful if the ulcer and parts beneath it have been cleanly excised in one block so that the freshly exposed tissues are unsoiled and their surface dry. Should the reverse be the case, the area may be treated with Dakin's solution or other antiseptic dressings until it has granulated, when a delayed graft is made.

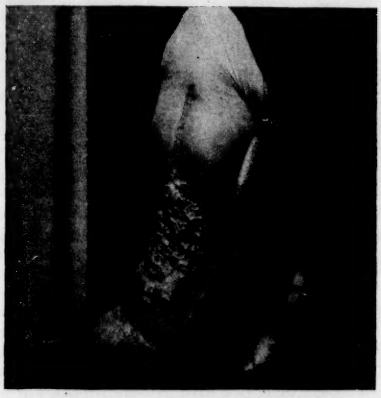

FIG. 4. E. S. Surg. No. 19407, Peter Bent Brigham Hospital. Post-phlebitic varix. The patient had suffered from ulcer for many years. One year before, the ulcer had been excised and skin-grafted. Subsequently the skin about the graft had broken down and the whole lower leg had become infected—the foot immensely swollen. Notice incisions and skin-grafts. Great areas of deep fascia have been removed.

This treatment will cure many cases but the patient should be told that more may be required. The leg may appear soundly healed, and yet, when the patient resumes an active life, breaking down of tissue about the graft, but rarely of the graft itself, may occur. In that case removal of more tissue is indicated and it is here that excision of strips of deep fascia, above, below and beside the grafted area may be undertaken. I show you a picture of a leg treated in this way. (Figures 4 and 5.) Before the final operation was performed the graft was surrounded by a zone of ulcer, the lower leg was of a universal brawny induration, the foot was swollen to twice its normal size,

its skin red and threatening to break down. Amputation was distinctly suggested. Incisions were made from ankle nearly to knee on the inner and outer sides of the calf and at several points above and below the ulcer. Wide strips of enormously thickened deep fascia were excised and the wounds allowed to gape. Secondary skin grafts were made some two weeks later with the result you see. Justification for this procedure lies not so much in the present condition of the region of the ulcer, which is excellent enough, but in the vastly improved condition of the foot.

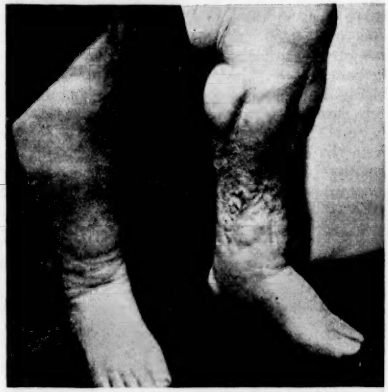

FIG. 5. E. S. Surg. No. 19407, Peter Bent Brigham Hospital. Post-phlebitic ulcer. Another view of leg shown in Fig. 4. Notice absence of swelling of foot.

Such are the principles upon which these post-phlebitic ulcerative conditions should, according to my experience, be treated. They are, by less radical methods, incurable and render their victims semi-invalids. Their operative treatment is looked upon by most physicians as a hopeless affair. Indeed, such treatment more than shares the bad name which operations for varicose veins have in the past so needlessly acquired. Yet varicose ulcers are as a rule susceptible of cure or of vast amelioration by surgery.

Most ulcers dependent upon simple varix are healed by rationally complete removal of the great saphenous system of veins. Ulcers of a post-phlebitic sort must be excised as if they were malignant tumors, and, in addition, an attempt should be made, when the indications suggest the necessity of it, to restore lymphatic drainage in the superficial tissues of the leg.

THE IMPORTANCE AND UNIMPORTANCE OF THE MURMUR*

BY WILLIAM H. ROBEY, M.D., BOSTON

A GENERATION ago the hearing of an abnormal sound in the heart was often the signal for a diagnosis of cardiac disease and usually an unfavorable prognosis. In those days cardiology was little understood and any unusual sound was looked upon with suspicion and dread. It colored the minds of medical teachers who consequently gave their students an exaggerated idea of the murmur with the result that some practitioners who received their training three or four decades ago have never been able to free themselves from an undue sense of its importance. It is still not uncommon to find a doctor who will make a diagnosis of organic heart disease if he hears a systolic murmur, no matter how lacking the patient may be in etiological factors or other cardiac and circulatory signs, while on the other hand he will pronounce a heart normal if without a murmur even though the patient has dyspnea upon ordinary exertion or is roused from sleep by precordial pain.

The question of the importance and unimportance of the mitral systolic murmur became prominent during the war when recruits were discovered to have this murmur during the course of a routine physical examination. Sir James Mackenzie returned thousands to the front who carried on successfully. During the early months of our participation in the war soldiers were sent into our Base Hospitals because a regimental surgeon had discovered a mitral systolic murmur. The question was whether the murmur was organic or functional. Was the murmur indicative of some organic lesion which would declare itself under the stress of action? We went into the history with great care and looked for other circulatory evidence. If the findings were negative save for the murmur, we made a diagnosis of functional murmur and signed the soldier for duty. The ruling of the Surgeon General was that if no other evidence of cardiac disease could be found the murmur was to be disregarded.

*Read before the North Berkshire Medical Society, January 8, 1924.

THE FUNCTIONAL SYSTOLIC MURMUR

Organic disease of the tissues which form the structure of the valves produces abnormal sounds or murmurs timed according to that por-

tion of the heart which by its contraction forces the blood through a constricted opening or back again from whence it came through an imperfectly closed aperture. While the great majority of functional murmurs are systolic in time, it is possible for any other murmur usually associated with organic disease to be functional. A very small number of functional murmurs are diastolic in time. While the belief is held by some that the majority of functional murmurs are caused by a temporary or permanent dilatation of the conus arteriosus, it is conceivable that any portion of the heart may be temporarily dilated or altered so that an abnormal sound is produced. Cabot gives the characteristics of functional murmurs as follows:

1. They are generally systolic and are usually heard with maximum intensity over the pulmonic valve area in the second left intercostal space and are transmitted in all directions and may be heard in the mitral area or aortic area sometimes with greater intensity than in the pulmonic.

2. Usually there is no enlargement of the heart nor accentuation of the pulmonic second sound.

3. They are often louder at the end of inspiration.

4. They are usually heard over a limited area, but there are exceptions.

5. They are quite variable in character; much more so than organic murmurs.

6. They are supposed to occur in anemic patients, yet in the severe anemias they are often absent, while they are found in robust and healthy individuals.

It is said that organic murmurs may occupy any part of the cardiac cycle. If systolic, they are often transmitted to the great vessels of the neck; there are changes in the second sounds and usually cardiac enlargement with signs of stasis in other organs. They are rarely loudest at the pulmonic area and are relatively uninfluenced by respiration, position or exercise.

The exceptions, however, connected with most of the statements have led to much controversy and have left the medical mind in doubt. Mackenzie was one of the first to urge the determination of this point because he felt that we were doing a great injustice to patients by stamping them victims of heart disease merely on the evidence of a murmur,—simply on the aural evidence of a sound which was not understood and because there existed something which varied slightly from the normal. In this way a normal healthy child could be transformed into a hopeless mollycoddle with a blasted career. How then shall the practitioner decide whether a murmur is to be regarded as important or unimportant? He can decide by applying the rules given above, but there are certain important additions which I would like to make and which should never be omitted in forming an opinion.

For the purpose of discussion we will use the term mitral regurgitation although in children and young adults it is doubtful if that condition ever existed per se. With the above as a premise we can state that no organic murmur, with the exception of mitral regurgitation, is without some evidence outside of the heart. That evidence is to be collected by a thorough history, by observation and a study of other organs and the circulation. It is a good plan for the beginner to form the habit of observing the patient's color, his breathing, the character of the superficial veins and arteries, the size of the palpable organs, the position of the cardiac impulse and the pulse pressure. If these are done the physician may make a correct guess as to what he will hear and then using his stethoscope will have a ready explanation for most murmurs. If he listens to the heart first he should have no opinion as to whether a murmur is functional or organic until he had applied the other tests. From an experience with graduate students I know that there are still busy practitioners who decide for or against organic heart disease upon the presence or absence of murmurs. Having applied all the tests you will go a long way towards a correct opinion.

But, you say, having applied all of these tests and found them negative, what about the persistent systolic murmur at the apex? Twenty years ago I attended a man of thirty, whom I had not known before, with a typical rheumatic fever. During the course of the fever, I discovered a mitral systolic murmur and with such an etiology I had a right to suppose that he had a rheumatic endocarditis. I did not make a positive diagnosis, although we were not so certain in those days that mitral regurgitation did not exist without mitral stenosis as a result of acute infectious diseases. He made a good recovery from rheumatic fever but the murmur remained. Of course, it is possible that he may have had a mitral systolic murmur before the onset of the fever, but I had no way of knowing. During the twenty years since he has not had a recurrence of the fever and I have examined him once or twice a year. No other cardiac signs developed and for the last eight years the murmur has not been heard. During this entire period he has worked very hard, sometimes doing two men's work. If a systolic murmur is heard during the course of an infection, it should be carefully noted and subsequent examinations made during the illness and at regular intervals afterwards. Frequent examinations of the heart are very necessary because the discovery of a pericardial friction rub during the height of the infection would suggest a pancarditis and would make a mitral systolic murmur much more important. In such a case the appearance of a mid-diastolic

murmur would strongly point to the significance of the systolic murmur—a mitral stenosis. Later the typical presystolic murmur might appear. In any event, the physician should not be in too much haste to form any opinion about damage done to the heart while the acute infection is in progress. Whatever is left during the convalescence and within the succeeding months, if it be a systolic or diastolic murmur for instance, the murmur should be studied with reference to all other cardiac and circulatory signs and effort tolerance. Some assistance in forming an opinion can be derived from the electrocardiograph, which may show muscle or bundle disturbances and thus aid in separating a functional from an organic murmur.

A girl of seventeen was seen January 20th, 1923, with a history of always being delicate. During the summer she had lost twelve pounds, although she was on a farm. She gave a history of rather frequent colds but no rheumatic fever, sore throats nor tonsillitis. Occasionally there was nose bleed. She had a good appetite and slept well, her chief complaint was very easy fatigue. If she went out for an evening she would be tired the following day. She was thin and of poor color. The blood pressure was 130-90. The heart was normal to percussion and fluoroscope. There was a loud blowing systolic murmur heard over the entire precordia and in the back. The impulse was heaving and the cardiac rate 120. There was no arrhythmia, no diastolic nor presystolic murmur, no thyroid enlargement, no cardiac thrill, no tremor of hands, no incoördination. The lungs and abdomen were negative. There was no edema. The hands were cold and slightly cyanotic. In the differential diagnosis, I considered first mitral stenosis and then effort syndrome and hyperthyroidism. In view of the fatigue, occasional nose bleeds and marked systolic murmur, the mitral stenosis seemed most probable. She was given a regime of rest and quiet with instructions to report in two weeks, but six days after I saw her, her mother reported that her daughter had a paralysis of the left arm and leg. This clarified the diagnosis of mitral stenosis. On March 23rd, 1923, I saw her in consultation with Dr. Casey and found, in addition to the hemiplegia, a systolic murmur, diastolic and presystolic murmurs and a marked apical thrill. The pulse was small and 140 in rate.

The history of a marked infection, the recurrence of tonsillitis or attacks of sore throat, would give greater significance to a mitral systolic murmur. Occasional nose bleeds or an arrhythmia (exclusive of sinus arrhythmia) in the presence of a mitral systolic murmur should suggest a probable mitral stenosis in a young person.

According to Cabot functional diastolic murmurs are very rare, but occur oftenest in conditions of profound anemia, when the hemoglobin is twenty-five per cent. or less, and of hypertension. Diastolic murmurs in mitral stenosis and aortic regurgitation can usually be differentiated by their time in the cardiac cycle and by the character of the pulsations in the peripheral vessels. The diastolic murmur of mitral stenosis is usually heard only over a very small area at the mitral area. The second sound is generally clearly audible, to be followed after an instant of time by the diastolic murmur. The diastolic murmur of aortic regurgitation is usually located at the left of the sternum, is more blowing in character and as a rule obscures the second sound. The Corrigan pulse is characteristic of aortic regurgitation while the pulse of mitral stenosis is of small volume and low tension. A combination of these two lesions, of course, alters the characteristics of the pulse of each. Sometimes the pulse of hyperthyroidism simulates the Corrigan, but on the gravity test it never gives the rapid fall. Arrhythmia is common in mitral stenosis, but comparatively rare in aortic regurgitation. Auricular fibrillation is a frequent occurrence in the former, but rarely occurs in the latter. Cabot and Locke (Johns Hopkins Hospital Bulletin, May, 1903) in their study of the clinical records of the Massachusetts General Hospital, 153 cases with autopsies, to determine the correctness of the clinical diagnosis, found mistakes were most frequent in tricuspid regurgitation and least frequent in aortic regurgitaton. They reported four cases with a diastolic murmur over the area where the murmur of aortic regurgitation is usually best heard, but at autopsy the valves and cavities were normal. All had visible pulsations or a pulse of the Corrigan type and with the diastolic murmur a diagnosis of aortic regurgitation was made. Three had marked anemia with low red counts and a hemoglobin ranging from 15 to 25 per cent. and since in severe anemias and in overaction of the heart visible pulsations occur in the carotid, subclavian, axillary, brachial, radial arteries and in the large arterial trunks of the lower extremities, it is probable that the pulsation simulated the water hammer pulse. In any event, the murmur was functional. You must have true aortic regurgitation to give the rapid collapse of the pulse, especially when aided by gravity.

CARDIO-RESPIRATORY MURMURS

Cardio-respiratory murmurs are most commonly heard along the left border of the heart and at the apex. They are usually systolic in time but may be diastolic. They may be due to a normal overlapping of the lung or to old adhesions between the pleura and pericardium. I have in mind several cases where there was an old history of pneumonia or phthisis on the left. The murmur varies with position and respiration. It is particularly strong during

inspiration. It is stated that the murmur will entirely disappear when the breath is held. This is not always the case. When the murmur is due to adhesions between the pericardium and pleura the movement of the heart will still produce the murmur, even when the lung is at rest. Recently we have seen such an example in the hospital. Anything which increases respiratory action usually strengthens the murmur, while true organic murmurs are little if any affected in this way. Pressure on the chest in the vicinity of the murmur diminishes its intensity, while organic murmurs are unaffected. The cardio-respiratory murmur is without accompanying objective or subjective symptoms and is of no importance. The murmur of tricuspid regurgitation is systolic in time but the diagnosis cannot be made unless we find a systolic venous pulsation in the jugulars and the liver, intense cyanosis with ascites and edema of the legs. You may be able to make out an area of dullness beyond the right sternal margin due to engorgement of the right auricle. I have not seen a case of tricuspid stenosis to recognize it. The diagnosis of aortic stenosis is quite frequently made, and on investigation, apparently from hearing a systolic murmur of greatest intensity in the aortic valve area. The murmur may or may not be transmitted into the vessels of the neck. It is one of the best examples of attempting to make a diagnosis on a murmur alone. One of the most striking cases of this occurred on the service of an able colleague. Some years ago when looking for a case of aortic stenosis to show the students I was told by the house physician that there was a case in Ward M. The patient, a man aged twenty-seven years, was convalescent from an acute multiple arthritis. He looked well and said that he was absolutely free of his discomforts. On listening to his heart he was found to have over the second interspace to the right of the sternum, a rough, grating murmur, systolic in time. On listening carefully, however, a similar murmur, very faint, was heard in diastole. There were absolutely no cardiac nor circulatory signs indicating aortic stenosis or regurgitation. In the absence of these confirmatory signs of valvular disease, outside of the precordia and taking into account the history of recent rheumatism, it seemed more probable that he had a pericardial friction rub. Two days later the rough sound, with its fainter diastolic, was heard over the third interspace to the left of the sternum, the following day over the fourth, and by the fifth day it was barely audible just above the cardiac impulse, and then disappeared. The chief difficulty seems to be in distinguishing the rub from a valve murmur. The rub, even when faint, will generally have a jerking, grinding or leathery quality. Because of the more active movements of systole it may seem to be wholly a systolic sound, but by listening carefully a sound of similar quality, sometimes very faint, will be heard in diastole. The rub may be mistaken for a valve murmur even by the most experienced. Within two months a consultation was held at the hospital to decide whether a patient had double aortic disease or pericarditis. The same considerations decided in favor of pericarditis. The rub often moves, from day to day, and is sometimes made more intense by pressure with the stethoscope. When the rub disappears it may mean that the plastic pericarditis is quieting down or fluid is accumulating.

In the first place, aortic stenosis, like mitral stenosis, rarely exists without its accompanying regurgitation. If you think of the mechanics of the lesion the reason is quite plain. I have seen two cases which I supposed were pure aortic stenosis, but probably the signs of regurgitation were not evident. The murmur is systolic in time, generally transmitted to the vessels of the neck, and often rough. It is accompanied by a palpable thrill. The pulse is of the plateau or sustained type. The second sound is absent or diminished, but Cabot reports that he has seen at autopsy two cases of aortic stenosis in which the aortic second sound was loud in life. It is the soft, blowing systolic murmur loudest at the aortic area, heard in middle-aged and elderly people with arteriosclerosis, which often causes a diagnosis of stenosis.

I heard a short time ago of an old man who had died of mitral regurgitation. A considerable number of old people have a systolic murmur at the apex, but they are actually suffering from myocardial degeneration. When they die it is of muscle failure and not because of a relative leak at the mitral valve.

There is no treatment for a murmur. In the earlier years of my practice I saw numerous cases in which digitalis was given merely on the evidence of a murmur, but in recent times this has become much less. A compensated heart with its particular disability clearly diagnosed rarely needs any treatment other than a well managed order of living. If the diagnosis is made early and the treatment of causes effected, the patient may go on for years without requiring any cardiac medication. Digitalis is for muscle disturbances with their accompanying arrhythmias. Unfortunately for us when the stage is reached where much attention to the direct treatment of the heart is necessary, the amount of help which we are able to give becomes progressively limited. Digitalis may be given, however, in certain cases of poor muscle tone with normal rhythm, and even normal rate, in small doses and such has been my custom for many years.

IS QUINIDINE OF VALUE IN THE TREATMENT OF AURICULAR FIBRILLATION?

Some Observations in Relation to the Vital Capacity of the Lungs*

BY S. A. LEVINE, M.D., AND ALBERT WILMAERS, M.D.†

In the past few years numerous reports of the results of the use of quinidine sulphate in the treatment of auricular fibrillation have appeared from various parts of the world. It is not our purpose here to review the literature on the subject but rather to place on record our experiences. Considerable enthusiasm has been expressed by many, as would naturally be expected of a new drug that promised help in the treatment of any chronic disease. It has occurred to us from the start that much of the enthusiasm was unwarranted, both from a theoretical point of view and now as a result of studying thirty-eight cases of auricular fibrillation treated in the Peter Bent Brigham Hospital.

The cardinal symptom of most patients suffering from heart disease is dyspnoea, and where auricular fibrillation is an added feature in the case, palpitation as a result of the rapid irregular heart action is also a frequent complaint. The mechanism of these two symptoms is different and has different origins. Dyspnoea improves or grows worse as the efficiency of the circulation changes. Palpitation on the other hand depends on the nervous state of the patient, whether the heart is regular or irregular, the actual rate of the heart and other factors. Palpitation may exist when the circulation is satisfactory or there may be no palpitation even when dyspnoea is extreme. It therefore follows that in judging any improvement of the efficiency of the circulation one should follow particularly the changes that indicate improvement in the signs of congestive failure and especially the complaint of dyspnoea. Clinically, the vital capacity of the lungs varies quite closely with the improvement in the signs of congestive heart failure and therefore such determinations were made in this study with the hope of finding out what value quinidine sulphate might have in patients with auricular fibrillation.

A study of thirty-eight patients was made of whom thirty-seven had persistent auricular fibrillation and one had auricular flutter. There were sixteen males and twenty-two were females. The ages ranged from fifteen to seventy-eight. There were two general groups, those with mitral stenosis of which there were twenty-three and those with chronic myocarditis without valve disease of which there were fifteen. Most of the patients gave evidence of more or less congestive heart failure although some were almost free from all symptoms and were admitted to the wards with the purpose of trying the effect of quinidine.

All the patients were put to bed and kept under careful observation. They were treated in the customary way, being given digitalis in full doses unless they were already well compensated or well digitalized. The purpose was to get whatever improvement was possible by all the other means available and then to try quinidine sulphate in order to be able to decide that if an improvement occurred it was due to the quinidine and not to the other measures used.

The quinidine was usually administered in the following way. First 0.2 gram was given twice a day to test the susceptibility of the patient. The following day either 0.3 or 0.4 gram was given three times a day, and each day the dose was increased 0.1 gram until the rhythm became regular or until it was thought wise to stop the drug. In this way patients receivd 0.4 gram, 0.5 gram, 0.6 gram and 0.7 gram three times a day on succeeding days. We did not go any higher than this latter figure. When a regular rhythm occurred we generally kept the patient on a maintenance dose of 0.1 gram three times a day. Many variations were necessary in the mode of administration of the drug. Often it had to be discontinued because of the development of distressing symptoms. The details of the administration and the effects produced are summarized in Table 1. Some patients were in such satisfactory conditions that no digitalis was given in their preparation, others received small amounts and still others were completely digitalized. In a few, digitalis was continued while quinidine was being given. Vital capacity readings were made by means of a water spirometer at frequent intervals, before, during, and after the digitalis therapy and then in relation to the administration of quinidine.

It is impracticable to publish all the vital capacity readings made on the patients and so it was decided to chart the readings at entry, just before and after quinidine was administered and at discharge from the hospital. Finally, a statement is made that summarizes our impressions as to whether the drug improved the condition of the patient or not. This statement reviews all the clinical factors both objective and subjective that might have any relation to changes in the state of the circulation, especially the vital capacity of the lungs.

Discussion

The administration of quinidine sulphate changed the mechanism of the heart in thirteen

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out of thirty-eight cases from auricular fibrillation to a regular rhythm. The duration of the normal mechanism was from one day to nine months. There were five patients who changed from auricular fibrillation to auricular flutter of whom two became regular and three returned to auricular fibrillation. The duration of the flutter was from less than a day to thirty-three days. All changes in rhythm were controlled by electrocardiographic tracings. There were three fatalities. One of these patients was in the stage of extreme cardiac failure and had not improved with the ordinary medical treatment. The other two fatalities were very well compensated when quinidine was started. It was known that one had been fibrillating for only three weeks and

TABLE 1

Case No.	Maximal dose of Quinidine in grams	Total amount of Quinidine	Regular, Yes or No	Flutter +	Duration of regular rhythm in days	Vital Capacity on admission in c.c.	Vital Capacity before Quinidine	Vital Capacity after Quinidine	Vital Capacity on discharge	Effect of Quinidine on Vital Capacity
				Chronic Myocarditis—Males						
1	0.3 t.i.d.	1.6	Yes		4	2400	2300	2650	2400	Increase
2	0.4 t.i.d.	2.8	"		56	3800	3800	3725	4200	No change
3	0.4 t.i.d.	6.4	No	+	33	3400	3400	3000	4050	Decrease
4	0.3 t.i.d.	13.6	"			2400	2400	2000	2450	"
	0.8 t.i.d.	12.6	"			2250	2200	2250	2250	No change
	0.7 t.i.d.	7.5	"			2100	2300	2300	2350	"
5	0.6 t.i.d.	9.4	"			2300	—	—	2400	?
6	0.6 t.i.d.	2.2	Yes		6	—	—	—	—	?
7	0.6 t.i.d.	2.9	"	+	18	4100	4100	3500	4100	Decrease
8	0.7 t.i.d.	9.5	No			2000	1900	1750	1950	"
				Chronic Myocarditis—Females						
9	0.6 t.i.d.	4.2	Yes		8	1800	1900	1850	1700	No change
10	0.7 t.i.d.	8.7	No			800	800	800	1125	"
11	0.7 t.i.d.	9.3	"	+	31	1000	950	1000	—	"
	0.6 t.i.d.	2.5	"	+		—	1250	1150	1800	"
12	0.5 t.i.d.	4.2	Yes		1	1300	1300	1400	1800	"
13	0.6 t.i.d.	3.9	"		2	850	1550	1800	1800	Increase
14	0.5 t.i.d.	3.1	No			1350	—	—	—	?
15	0.3 t.i.d.	1.7	Yes		1	—	—	—	—	?
				Valvular—Males						
16	0.4 t.i.d.	2.8	No			2600	2600	3600	—	Increase
	0.4 t.i.d.	4.4	"			—	3900	4400	—	"
	0.4 t.i.d.	7.2	"			—	4650	4650	4650	No change
	0.7 t.i.d.	8.3	"			2650	—	4450	4250	?
17	0.3 t.i.d.	2.8	Yes		28	4400	4450	4500	4600	No change
18	0.6 t.i.d.	13.0	No			2500	2500	2800	2750	Increase
19	0.7 t.i.d.	7.7	Yes		47	3250	3700	—	—	?
20	0.8 t.i.d.	7.8	No			1800	1350	1350	1800	No change
21	0.4 t.i.d.	1.9	"			1800	2000	2400	2500	Increase
22	0.5 t.i.d.	6.7	"			1900	1850	1850	2000	No change
23	0.3 t.i.d.	1.0	"			1800	1800	1800	1900	"
				Valvular—Females						
24	0.4 b.d.	1.2	No			1300	1950	2100	—	No change
	0.4 b.d.	1.8	"	+	2	—	2100	2150	2400	"
25	0.3 t.i.d.	9.4	"			800	1050	1050	1050	"
26	0.3 t.i.d.	24.4	"			1200	1200	1800	—	Increase
	0.6 t.i.d.	3.0	Yes		32	—	—	1700	1850	?
	0.6 t.i.d.	7.0	"		26		1600	1500	1750	No change
27	0.6 t.i.d.	5.6	No			1200	—	—	—	?
	0.6 t.i.d.	5.8	"			—	2100	2250	2300	No change
28	0.6 t.i.d.	2.0	"			1200	1200	—	950	?
29	0.6 t.i.d.	9.4	"			1800	2300	2300	2350	No change
30	0.4 t.i.d.	8.4	"			—	—	—	—	?
	0.5 t.i.d.	3.3	"			—	—	—	—	?
31	0.6 t.i.d.	4.9	"			1500	2150	1800	1800	Decrease
32	0.6 t.i.d.	3.4	Yes	+	270	—	—	—	—	?
33	0.7 t.i.d.	10.0	No			2250	2300	2600	2700	Increase
34	0.7 b.d.	4.8	"			2300	2300	2600	—	"
	0.5 b.d.	2.3	"			—	2500	2400	2300	No change
35	0.3 t.i.d.	1.3	Yes		13	975	1150	1500	1600	Increase
36	0.4 t.i.d.	2.5	No			1700	—	—	—	Died
37	0.2 b.d.	0.7	"			2000	—	—	—	"
	0.3 b.d.					—	—	—	—	
38	0.2 b.d.	1.0	Yes		2	1400	1950	—	—	"

had not been much incapacitated. In fact, this patient had been working every day and came in to be prepared for a tonsillectomy. The heart at autopsy showed mitral stenosis and only weighed 300 grams. In both of these latter fatal cases the ordinary prognosis that would have been given if the patients were treated with digitalis and the more usual procedures would have been 5 to 10 or more years. Post-Mortem examination in each of these three cases showed no thromboses in the heart or any evidence of emboli. Nothing but a toxic phenomenon could be given as the cause of death.* It must be appreciated that in order to warrant using a drug that shortened the lives of these two patients by many years, such a procedure must promise considerable improvement in those that survive.

Let us consider the group of patients who were made regular. There were 13 such patients but in only four did the regular rhythm last more than one month. In all cases that could be followed, fibrillation eventually returned although in one patient the normal mechanism persisted for nine months. In no case was there a very striking increase in the vital capacity of the lungs when the heart became regular. Frequently there was no appreciable change, occasionally it would be lower while regular than while the heart was fibrillating and at other times there would be a slight but definite increase on becoming regular with a corresponding decrease as fibrillation recurred. Of all these patients there was only one whose improvement in general health was considerable and enough to warrant taking the risk of immediate mortality that always accompanies the use of the drug and even this subjective improvement only lasted nine months.

When the regular rhythm was not restored, the effect of the drug, although temporary, was not beneficial. The heart rate would rise, the patient would have more palpitation and occasionally there would be nausea, vomiting or ringing in the ears. These symptoms would always disappear on omitting the drug. When auricular flutter resulted from quinidine therapy the condition of the patient was on the whole worse, even though the heart might thereby become regular. The ventricular rate would be faster and although in all instances the flutter eventually changed to normal rhythm or back to fibrillation, it might have been impossible to alter the flutter.

Our experience with one case throws an interesting side light on the use of quinidine. This patient had mitral stenosis and auricular fibrillation and responded favorably to digitalis and the ordinary measures used in the treatment of heart failure. She became ambulatory, but nine months later her heart was regularized by quinidine in another hospital. A short time after this, severe symptoms of circulatory insufficiency recurred and then when the heart was rapid and regular no improvement could be obtained with digitalis and the patient died. It seemed to us that if auricular fibrillation had persisted the digitalis would have been more helpful and might well have restored the circulation to some extent. For it is well known that a rapid heart rate slows more readily as a result of digitalis therapy if auricular fibrillation exists than when the rhythm is normal.

From these cases it seems that quinidine cannot offer any help in the treatment of the average hospital patient with auricular fibrillation who has had evidence of congestive heart failure, even if given digitalis and the customary bed treatment is employed to the stage where signs of failure have disappeared.

The majority of such patients fail to become regular under quinidine therapy and of those that do respond only a very few remain so for more than a month. There are occasional sudden deaths that result from the drug even if the heart has been fibrillating for only a very short time and when the patient's circulation is in a most satisfactory condition. There is no evidence that the circulation is improved when the heart is made regular, for the essential factor is the rate of contraction of the ventricles and not the rhythm. When a fibrillating heart can be kept fairly slow by digitalis the circulation often proves to be as efficient or more so than when the heart is regular, for often the ventricular rate if regular is more rapid when digitalized than if the auricles are fibrillating. Palpitation is the one complaint that may be helped by making the heart regular. There is no reliable evidence that the shortness of breath or the length of life of the individual would be improved by the use of quinidine.

One theoretical feature of this problem must be considered. If auricular thrombi are more apt to form in auricles that are fibrillating than those that are contracting regularly, it may prove to be true that emboli either into the pulmonary or general systemic circulation will occur less commonly in patients that respond favorably to quinidine than those who persist with fibrillating auricles. This point cannot at present be answered. It will take many years to accumulate sufficient data along this line, and in one sense this feature of the problem is not concerned with the question of immediate improvement of the circulation by the drug.

SUMMARY

1. Quinidine Sulphate was administered to thirty-seven hospital patients with auricular fibrillation, and one with auricular flutter.

2. Special attention was given to the changes in the vital capacity of the lungs as a result of the use of this drug.

3. The rhythm of the heart became regular in

*Complete pathologic reports of the three fatal cases are to be reported later.

thirteen cases, auricular flutter was observed in five, and there were three unexpected fatalities. In only four did the regular rhythm last more than one month, and in none more than nine months.

4. No important changes in the vital capacity of the lungs were found that could be attributed to quinidine.

5. It follows from this study that quinidine sulphate is of no practical value in the treatment of patient with auricular fibrillation who previously had congestive heart failure. The more conservative treatment with digitalis is the more satisfactory.

6. The use of quinidine should not become general but should be confined to a few who might still be making careful observations as to its proper place in the treatment of heart disorders.

CHRONIC MYOCARDITIS—MALES

Case 1. Medical No. 16575—Age 64—markedly decompensated—digitalis given before but not during quinidinization.

Case 2. Medical No. 16443—Age 59—markedly decompensated—digitalis given before but not during quinidinization.

Case 3. Medical No. 16522—Age 50—markedly decompensated—digitalis given before but not during quinidinization.

Case 4. Medical No. 16586—Age 57—markedly decompensated—digitalis given before and during quinidinization.

Case 4. Medical No. 18193—Age 57—markedly decompensated—digitalis given before but not during quinidinization.

Case 4. Medical No. 19765—Age 57—markedly decompensated—digitalis given before but not during quinidinization.

Case 5. Medical No. 16919—Age 72—markedly decompensated—digitalis given before and during quinidinization.

Case 6. Medical No. 16952—Age 78—markedly decompensated—digitalis given before but not during quinidinization.

Case 7. Medical No. 17702—Age 50—slightly decompensated—digitalis given before but not during quinidinization.

Case 8. Medical No. 18305—Age 67—markedly decompensated—digitalis given before but not during quinidinization.

CHRONIC MYOCARDITIS—FEMALES

Case 9. Medical No. 16745—Age 67—slightly decompensated—digitalis not given before nor during quinidinization.

Case 10. Medical No. 18023—Age 60—markedly decompensated—digitalis given before and during quinidinization.

Case 11. Medical No. 17972—Age 65—markedly decompensated—digitalis given before and during quinidinization.

Case 11. Medical No. 17972—Age 65—markedly decompensated—digitalis given before but not during quinidinization.

Case 12. Medical No. 17943—Age 47—slightly decompensated—digitalis given before but not during quinidinization.

Case 13. Medical No. 18701—Age 75—markedly decompensated—digitalis given before but not during quinidinization.

Case 14. Medical No. 18974—Age 43—slightly decompensated—digitalis not given before but during quinidinization.

Case 15. Medical No. 19889—Age 65—slightly decompensated—digitalis given before but not during quinidinization.

Case 16. Medical No. 16292—Age 43—markedly decompensated—digitalis given before but not during quinidinization, on two different occasions; at another time digitalis given both before and during quinidinization.

Case 16. Medical No. 18373—Age 44—markedly decompensated—digitalis given before but not during quinidinization.

Case 17. Medical No. 16620—Age 34—no decompensation—digitalis not given before nor during quinidinization.

Case 18. Medical No. 16908—Age 42—markedly decompensated—digitalis given before but not during quinidinization.

Case 19. Medical No. 17998—Age 42—slightly decompensated—digitalis not given before nor during quinidinization.

Case 20. Medical No. 18194—Age 19—slightly decompensated—digitalis given before but not during quinidinization.

Case 21. Medical No. 18924—Age 45—slightly decompensated—digitalis given before but not during quinidinization.

Case 22. Medical No. 19712—Age 43—markedly decompensated—digitalis given before but not during quinidinization.

Case 23. Medical No. 20144—Age 50—markedly decompensated—digitalis given before but not during quinidinization.

VALVULAR—FEMALES

Case 24. Medical No. 16150—Age 45—markedly decompensated—digitalis given before but not during quinidinization.

Case 24. Medical No. 16150—Age 45—slightly decompensated—digitalis not given before nor during quinidinization.

Case 25. Medical No. 16588—Age 53—markedly decompensated—digitalis given before and during quinidinization.

Case 26. Medical No. 16623—Age 38—extremely decompensated—digitalis given before but not during quinidinization.

Case 26. Medical No. 16623—Age 38—markedly decompensated—digitalis given before but not during quinidinization.

Case 26. Medical No. 17870—Age 39—extremely decompensated—digitalis given before and during quinidinization.

Case 27. Medical No. 16950—Age 46—extremely decompensated—digitalis given before and during quinidinization.

Case 27. Medical No. 16950—Age 46—markedly decompensated—digitalis given before and during quinidinization.

Case 28. Medical No. 16964—Age 47—extremely decompensated—digitalis given before and during quinidinization.

Case 29. Medical No. 17047—Age 47—no decompensation—digitalis given before and during quinidinization.

Case 30. Medical No. 17483—Age 65—slightly decompensated—digitalis given before but not during quinidinization.

Case 30. Medical No. 17483—Age 65—no decompensation—digitalis given before and during quinidinization.

Case 31. Medical No. 18659—Age 39—markedly decompensated—digitalis given before but not during quinidinization.

Case 32. Medical No. 18923—Age 41—no decompensation—digitalis given before but not during quinidinization.

Case 33. Medical No. 19079—Age 15—slightly decompensated—digitalis given before but not during quinidinization.

Case 34. Medical No. 19108—Age 35—no decompensation—digitalis given before but not during quinidinization.

Case 34. Medical No. 19108—Age 35—no decompensation—digitalis not given before nor during quinidinization.

Case 35. Medical No. 19645—Age 15—markedly decompensated—digitalis given before but not during quinidinization.

Case 36. Medical No. 20142—Age 39—markedly decompensated—digitalis given before but not during quinidinization.

Case 37. Medical No. 20762—Age 26—extremely decompensated—digitalis given before but not during quinidinization.

Case 38. Medical No. 22389—Age 33—no decompensation—digitalis given before but not during quinidinization.

DIGITALIS AND REGULAR RHYTHM

BY CLIFTON B. LEECH, M.D.

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SINCE the publication of Robinson's[1] notable monograph there would be little excuse for additional papers dealing with digitalis therapy if it were not true that clinicians still are at variance in regard to indications for the drug. This is especially true, for the general practitioner at least, in those cases of heart disease which present normal rhythm. There is quite a general feeling that digitalis is of little value except for auricular fibrillation. This erroneous impression has been given prominence by Thomas Lewis[2]. It is not an uncommon practice for digitalis to be given to every sort of heart disturbance, functional and organic, without knowing what effect may be expected. There is considerable lack of uniformity of result because of marked differences in the dosage and the preparations of digitalis in use. Medical literature is filled with references concerning the use of digitalis in various diseases of the cardio-vascular system without giving in detail the method of therapy, the dose, and the precise effect. Confusion exists in the minds of men who find that former teachings in regard to the physiological action of digitalis are not accepted today in their entirety. Confidence in the medicine is lost when it is discovered that digitalis is not a powerful "tonic" which always restores strength and force to diseased heart muscle.

The purpose of this paper is to show the clinical effect of digitalis in cases presenting regular rhythm.

In each case cited the patient was digitalized, that is, digitalis was given to the maximum amount which the patient could take without toxic effect, as shown by the approach of nausea and by the effect upon the heart rate and upon the T wave of the electrocardiogram. After digitalization a daily ration was given sufficient to keep the patient just under saturation. This amount was equal to the daily excretion of digitalis, an average of .15 gramme. The approximate amount of digitalis required for saturation was pre-determined according to Eggleston's plan[3]. One quarter of this amount was given by mouth at six-hour intervals until three quarters of the total estimated quantity had been taken. Then one eighth of the total was given every four hours until there was evidence of saturation.

The digitalis preparation used in this series, with one exception, was the standardized powdered leaf in pills of .1 gramme. This preparation has the advantage of keeping indefinitely, in stoppered bottles, without losing potency. Pills are convenient for the patient and are not subject to inaccuracies of measurement by nurses or others. Pills of this strength also simplify the arithmetical procedure in determining the dosage. Thus: one pill for each 10 lbs. of body weight. Patient weighing 160 lbs. is estimated to require 16 pills. The dosage equals 4 pills every six hours for three doses, then 2 pills every four hours until saturation.

I. ARTERIO-SCLEROTIC HEART DISEASE, REGULAR RHYTHM

Case: Male, age 60 years, second admission for similar complaint.

Complaint: Shortness of breath, precordial pain, cough.

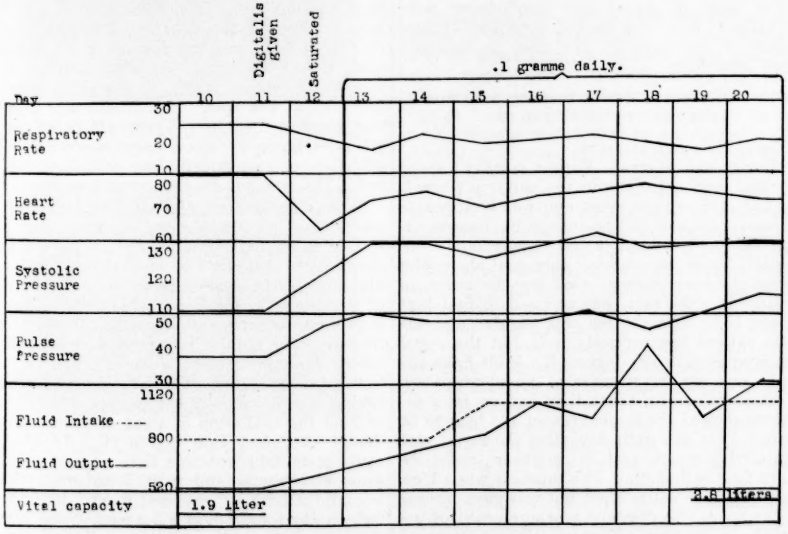
Past History: No pre-cardiac disease, life of hard work.

Physical Examination: Small, thin, old man weighing 134 lbs. Few carious teeth. Tonsils small and smooth. Arcs senilis, pupils normal, retinal vessels tortuous and sclerotic. Blood pressure 110-72 both arms. Congestion in the right lower lung, respiration 24. Heart moderately enlarged, no murmurs, rate 80, regular rhythm. No evidence of circulatory congestion in liver or extremities. Radial vessels markedly sclerotic. Radial pulses equal in volume. Urinary output about 500 c.c. Small trace of albumen, occasional cast, kidney function normal. Vital capacity 1.9 liters.

Progress: After 10 days in bed (unable to lie flat) there was no improvement in symptoms and signs. The average fluid intake was 800 c.c. while the average urinary output was 520 c.c. On the eleventh day the administration of digitalis began. On the twelfth day, after .8 gramme of digitalis had been given the patient became nauseated and the pulse rate dropped to 64 but remained regular. On the thirteenth day the heart rate was 72, respiration 16, the blood pressure 130-74 in the left arm and 120-70 in the right arm. Clinical improvement continued until the twentieth day when the patient was ambulatory. He was then symptom free, respirations were 18, heart rate 72. and the vital capacity 2.8 liters. The average daily urinary output for the final six days was 1000 c.c. which equalled the average fluid intake.

In a similar case of arterio-sclerotic heart disease which showed moderate congestive failure digitalization was accomplished four times, each time with marked effect. In this case the most striking effect, apart from improvement in symptoms, was an elevation in the systolic pressure from 15 to 30 millimeters of mercury. The diastolic pressure was unchanged or slightly lowered resulting in an increase in pulse pressure. This effect was not thought to be due to action upon the vessels themselves but to be a result of diminished stasis within peripheral vessels.

FIGURE I. EFFECT OF DIGITALIS IN CASE OF ARTERIO-SCLEROTIC HEART DISEASE



Eight cases of arterio-sclerotic heart disease were digitalized; in six cases there was definite clinical improvement, in two cases the digitalis produced no demonstrable effect.

II. MYOCARDIAL INSUFFICIENCY, REGULAR RHYTHM

Case: Male, age 55 years, ambulatory.

Complaint: "All gone" feeling in the chest and abdomen after very slight exertion. Marked dyspnea upon exertion.

Past History: No pre-cardiac disease, life of hard work. For the past six months has done no work but has not been confined to bed.

Physical Examination: Well nourished man, of middle age, weighing 165 lbs. No evidence

of heart enlargement by roentgenogram or fluoroscope. The heart sounds were faint, especially the first sound. No murmurs. No evidence of circulatory congestion. Heart rate 84, regular rhythm. Radial vessels soft. Radial pulses equal. Blood pressure 110-80 in right arm, 124-80 in the left arm. Vital capacity 2.4 liters. Average urinary output 1500 c.c.

Progress: This patient was not put to bed but was allowed to rest at home. During 48 hours he was given 1.9 gramme digitalis after which a daily ration of .2 gramme was administered. After six weeks his "all gone" sensations disappeared and moderate exertion produced no dyspnea or discomfort. The pulse rate, the blood pressure and the urinary output were not changed but the vital capacity increased to 3.6 liters.

Ten cases of myocardial insufficiency were digitalized; five cases showed apparent definite benefit of some sort, in five cases no improvement occurred.

III. RHEUMATIC HEART DISEASE, REGULAR RHYTHM, WITHOUT FAILURE

Case: Male, age 11 years.

History and progress: For six months a small boy had been a patient in the children's ward. He had a tubercular spine and was strapped to a frame. He had had no infectious diseases except chicken-pox and a very occasional sore throat. There was nothing abnormal about him except the bone disease. One day his nurse, in noting his pulse rate, was alarmed to find that it was 136. The rhythm was regular and the little patient felt perfectly well, but the rectal temperature was 100 degrees F. Aside from the rapid rate the heart revealed no abnormality. Electrocardiograms taken then and on later occasions showed the mechanism of the beat to be normal. On the sixth day after the rapid rate appeared a rough systolic murmur could be heard for the first time. This murmur was a blow heard over the entire chest but was most intense at the apex. No diastolic murmur could be detected at any time. There was no evidence of infection of the tonsils, teeth or sinuses. There had been no sore throat for several months. Kidney function and urine were normal.

After prolonged treatment with salicylates, the fever subsided but the heart rate remained about 130, and the systolic murmur was unchanged. The patient was still on the frame. The boy was then digitalized without effect upon the heart rate. The tonsils were finally removed. Four months later the heart rate was still very rapid, from 110 to 130. Again the patient was digitalized without effect. During all this time the patient presented no symptoms, and numerous electrocardiograms showed normal rhythm.

Six patients (five children—one adult) with rheumatic heart disease, regular rhythm, without failure were digitalized during and after acute endocardial infection. In no case was the heart rate affected by the drug.

IV. RHEUMATIC HEART DISEASE, MITRAL STENOSIS, REGULAR RHYTHM

Case: Female, age 7 years.

History and progress: A child with a negative past history except for tonsilitis underwent tonsillectomy. At the time of the operation the patient was normal in every respect except for hypertrophic and obstructive tonsils. Four months after the tonsillectomy and without discoverable exciting cause an acute endocardial inflammation occurred. For eight months the child was kept in bed in the hospital at the end of which time the acute infection had run its course and the patient presented all the evidence of mitral stenosis. She was thought to be a hopeless case. The heart was slightly enlarged. The rhythm was regular with a rate of 110 to 120. The precordium arose forcibly with each heart beat. A harsh systolic blow was heard over the entire chest with its greatest intensity at the apex. A mid-diastolic rumble was heard at the apex. There was no edema or other evidence of circulatory congestion, due probably to the complete, long continued rest in bed. The respiratory rate was 24 to 30.

It was decided to digitalize this patient in accordance with a suggestion by Cohn and Fraser as quoted by Robinson[1]. They pointed out that "a delay in the conduction of the cardiac impulse from auricles to ventricles may be of advantage to the heart when mitral stenosis is present, as such an effect would increase the time available for the left auricle to empty itself before the onset of ventricular contraction." In October, 1923, this patient was given, during a twelve day period, 2.9 grammes of digitalis without the exhibition of signs of saturation although the heart rate fell to 90. An electrocardiogram taken at this time showed marked sinus arrhythmia and a P R interval of .16 second. Digitalis was continued, .1 gramme twice a day for six days when nausea and vomiting occurred. An electrocardiogram now showed a prolonged P R interval of .24 second. Sinus arrhythmia was not so marked but the rate remained practically unchanged. At this time distinct clinical improvement was noted. The heart action was less labored, respirations were easier, and the patient appeared to be brighter. No change was detected in the heart sounds. Digitalis rations were continued for several months with similar good effect until the patient became ambulatory. Her prognosis as to life is limited but it is expected that she will have ten to fifteen years of comfortable restricted activity.

This is the only case of mitral stenosis in which the author has had proof of the effect of digitalis upon auricular conduction.

Four cases (three children, one adult) of rheumatic heart disease, mitral stenosis, conges-

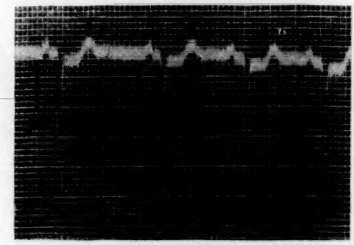
tive failure, regular rhythm have benefited by digitalization.

V. AORTIC REGURGITATION, REGULAR RHYTHM

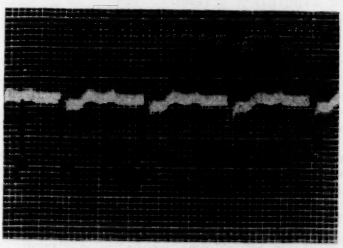
This form of valvular disease is thought by some to be a contra-indication to digitalis.

The author has never attempted to treat aortic regurgitation *per se* by digitalis but he has treated heart failure of various types in patients who have also presented this lesion.

FIG. 2. Effect of digitalis in case of mitral stenosis.



A. Lead II of electrocardiogram before digitalization was complete. P R interval (conduction time from sinus node to auriculo-ventricular node) is .16 sec. T is upright. Each time interval = .04 sec.



B. Lead II of electrocardiogram taken at complete digitalization. P R interval is .24 sec. T is nearly flat.

In eleven cases of aortic regurgitation which were digitalized there has been nothing to indicate that the drug should not be used in the presence of this condition.

VI. COMPLETE HEART BLOCK

In complete heart block the rhythm of the ventricular contractions is regular or very nearly so. In this condition, since no impulses of supra-ventricular origin are received by the ventricles, it is obvious that any effect produced by digitalis upon ventricular contraction must be a direct effect upon ventricular tissue rather than upon the vagus or conducting apparatus. In complete block it is obvious that there can be no contra-indication to digitalis.

Case: A male, 70 years of age, entered the hospital with severe congestive failure. There was considerable oedema of the legs, abdominal ascites, rales in both lungs, and a pulse rate of 30. The bradycardia was known to have existed for 20 years with an average rate of about 34, having never gone higher than 52 or lower than 28. There was no history of Stokes-Adams syndrome. Electrocardiograms showed complete heart block, auricular rate 150, ventricular rate 29.

The congestive failure did not improve after prolonged bed rest and saline purging. Tincture of digitalis was administered, 300 minims in ten days, during which time there was rapid and marked improvement. Digitalis rations were continued and in three weeks the patient left the hospital with no evidence of circulatory congestion. Heart block was unchanged, the ventricular rate remaining about 30.

VII. EFFECT OF SMALL DOSES

Small doses of digitalis are reputed to be of value in certain conditions. Daily administration of small amounts of digitalis is spoken of as a "tonic" dose. The adjective seems to apply to the reputed effect of digitalis in such dosage. In a private communication an eminent internist stated that he considered a daily dose of .1 gramme of digitalis leaf to be of distinct benefit to patients with relatively high diastolic blood pressure. He believed that the digitalis not only produced a lowerng of the diastolic pressure but also strengthened the heart muscle.

If, as Pardee[4] has shown, the average daily elimination of digitalis from the human body is about .15 gramme it is difficult to understand by what mechanism the "tonic" dose produces its effect or why such a dose should have a different and more beneficial effect than larger amounts. It would seem that any desirable effect of the drug might be increased by some degree of saturation.

Eighteen patients, presenting various diagnoses, but all showing relative increase in diastolic pressure, were studied. Each case, regardless of body weight, received .1 gramme of digitalis leaf daily for one month. Careful observations were made to determine any clinical result. Pressure readings were taken in both arms. The arm giving the higher reading was chosen for later comparison. A mean of three readings was recorded.

The results of this small series indicate that the "tonic" dose is without constant or definite value in the treatment of high diastolic blood pressure.

The systolic pressure was increased in nine cases, decreased in six cases, and unchanged in three cases. The average increase was 3 mm. of mercury, the average decrease was slightly more than 3 mm.

The pulse pressure was increased in ten cases, was diminished in seven cases, and unchanged in one case. The average increase was 3.6 mm. of mercury, the greatest increase was 11 mm. The average decrease was slightly more than 2 mm.

The pulse rate was elevated in four cases, was lowered in seven cases, and was unchanged in seven cases. The average elevation was 4 beats per minute, the average fall was 3.5 beats per minute.

The cases which were reported as feeling better presented no particular evidence of digitalis action, but it may be noted that of the four cases three were light in body weight. These may have become more or less saturated with the drug.

FIG. III

EFFECT OF SMALL DOSES OF DIGITALIS

No.	Rate		Systolic		Diastolic		Pulse Pressure		Diagnosis	Symptomatic improvement	Weight, lbs.	Age	Sex
	Before	After	Before	After	Before	After	Before	After					
1	94	94	136	134	98	98	38	36	Mitral stenosis	Feels better	128	35	Female
2	80	82	115	116	90	90	25	26	Mitral stenosis	None	140	42	Male
3	78	78	120	118	88	88	32	30	Mitral stenosis	None	137	39	Female
4	88	84	115	112	80	78	35	34	Angina pectoris	None	160	64	Male
5	96	100	134	137	102	103	32	34	Angina pectoris	None	124	45	Female
6	80	81	135	135	86	86	49	49	Angina pectoris	None	151	63	Male
7	78	72	180	185	117	111	63	74	Obesity—Hypertension	None	199	60	Female
8	85	83	179	180	120	120	59	60	Obesity—Hypertension	None	232	48	Female
9	72	70	166	161	108	100	58	61	Hypertension	None	130	49	Female
10	110	120	230	240	160	165	70	75	Chronic nephritis with hypertension	None	134	32	Female
11	76	70	150	150	96	98	54	52	Subject to attacks of parox. tachycardia	Feels better	133	50	Male
12	74	73	183	180	120	118	63	62	Hypertension	None	150	43	Male
13	90	86	190	190	135	140	55	50	Hypertension	None	139	41	Female
14	82	82	148	150	96	90	52	60	Myocardial insuff.	Feels better	144	57	Female
15	80	80	130	132	90	90	40	42	Myocardial insuff.	Feels better	163	50	Male
16	68	68	142	138	92	90	50	48	Myocardial insuff.	None	203	59	Male
17	92	92	118	120	88	88	30	32	Myocardial insuff.	None	152	49	Female
18	80	80	168	169	122	122	46	47	Arteriosclerosis	None	160	50	Male

SUMMARY

1. Digitalis is useful in many cases presenting regular rhythm especially if there is congestive circulatory failure. Patients should be digitalized.

2. Eight cases of arterio-sclerotic heart disease, regular rhythm, were digitalized—in six there was clinical improvement.

3. Ten cases of myocardial insufficiency were digitalized—in five cases there was clinical improvement.

4. In six cases of rheumatic heart disease, regular rhythm, without failure, there was no evidence that digitalization was of benefit.

5. Four cases of rheumatic heart disease, mitral stenosis, regular rhythm, congestive circulatory failure, were improved clinically by digitalization. In one case of mitral stenosis digitalis produced a depression of auricular conduction (prolonged P R interval) coincident with marked clinical improvement.

6. Aortic regurgitation does not contraindicate the use of digitalis.

7. In a case of complete heart block, congestive failure, digitalis produced clinical improvement. In this case the author considers that the digitalis effect was probably a direct effect upon the ventricular musculature.

8. In eighteen cases of relative high diastolic blood pressure there was no evidence of effect from small doses of digitalis. Small doses of digitalis produced no constant or marked change in pulse pressure, systolic pressure, and heart rate. There was no evidence that small doses of digitalis were of value in mitral stenosis, angina pectoris, hypertension, and myocardial insufficiency.

REFERENCES

1 Robinson: The Therapeutic Use of Digitalis. Medicine, pp. 1-137, May, 1922.
2 Lewis: Cardinal Principles in Cardiological Practice. Brit. Med. Jour., Vol. II, p. 621, 1919.
3 Eggleston: Administration of Digitalis by "Eggleston Method." Jour. Am. Med. Assn., Vol. LXXIV, p. 733, 1920.
4 Pardee: Notes on Digitalis Medication. Jour. Am. Med. Assn., Vol. LXXIII, p. 1822, 1919.

The Red Cross Societies stand ready to function in time of distress and disaster and are ever striving to solve some of the fundamental problems of national welfare which every country has to face. They represent the great humanitarian effort which has made their name respected the world over.—*Herbert Hoover.*

The Massachusetts Medical Society

PROCEEDINGS OF THE COUNCIL

STATED MEETING, FEBRUARY 4, 1925

A STATED MEETING of the Council was held in John Ware Hall, Boston Medical Library, February 4, 1925, at 12 o'clock, noon. The President, Dr. E. H. Bigelow, was in the chair and the following 105 Councilors present:

BARNSTABLE
W. D. Kinney

BRISTOL NORTH
W. H. Allen

BRISTOL SOUTH
R. W. Jackson
E. F. Cody

ESSEX NORTH
E. S. Bagnall
R. V. Baketel
J. Forrest Burnham
T. R. Healy
G. E. Kurth
F. W. Snow
W. D. Walker

ESSEX SOUTH
W. K. Foster
W. T. Hopkins
P. P. Johnson
G. M. Kline
A. N. Sargent

FRANKLIN
G. P. Twitchell

HAMPDEN
E. P. Bagg, Jr.
H. D. Gafney
E. A. Knowlton

HAMPSHIRE
E. D. Williams

MIDDLESEX EAST
A. E. Small
G. F. Dow
Richard Dutton
R. R. Stratton

MIDDLESEX NORTH
A. R. Gardner
W. B. Jackson
J. H. Lambert
J. A. Mehan
T. A. Stamas

MIDDLESEX SOUTH
E. H. Bigelow
A. H. Blake
F. B. M. Cady
W. H. Crosby
F. G. Curtis
C. E. Mongan
Dwight O'Hara
C. F. Painter
W. A. Putnam
W. D. Ruston
J. W. Sever
C. H. Staples
A. K. Stone
Fresenius Van Nuys
H. R. Webb
Alfred Worcester

NORFOLK
D. N. Blakely
H. K. Boutwell
W. L. Burrage
P. W. Carr
Samuel Crowell
C. G. Dewey
D. G. Eldridge
C. B. Faunce, Jr.
C. S. Francis
A. H. Hodgdon
G. W. Kaan
C. J. Kickham
Edward Martin
J. S. May
T. J. Murphy
M. V. Pierce
Victor Safford

NORFOLK SOUTH
C. S. Adams
O. H. Howe

PLYMOUTH
J. H. Lawrence
A. L. Beals
N. K. Noyes
Gilman Osgood

SUFFOLK
C. M. Smith
J. W. Bartol
Robert Bonney
V. Y. Bowditch
David Cheever
G. B. Fenwick
Channing Frothingham
J. E. Goldthwait
W. C. Howe
J. C. Hubbard
F. B. Lund
G. B. Magrath
T. J. O'Brien
R. B. Osgood
E. H. Place
Alexander Quackenboss
W. H. Robey
Jane D. K. Sabine
D. D. Scannell
J. S. Stone
Louisa P. Tingley

WORCESTER
L. R. Bragg
W. P. Bowers
W. J. Delahanty
G. A. Dix
G. E. Emery
M. F. Fallon
Homer Gage
David Harrower
E. L. Hunt
A. G. Hurd
A. W. Marsh
F. H. Washburn
S. B. Woodward

WORCESTER NORTH
W. E. Currier
A. F. Lowell

The record of the last meeting was read in abstract by the Secretary. There being no omissions or corrections noted the minutes were accepted as read and printed. The President made the following remarks:

The welfare of the Massachusetts Medical Society depends upon the prosperity of the District Societies. Any harm coming to one of our eighteen District Societies hurts its parent Society, whose children they are. Our Commonwealth is jealous for the well-being of the cities and towns created by the State. In like manner, the Massachusetts Medical Society has a vital interest in everything affecting the Districts from the Berkshires to the Cape. I find increased interest in the District meetings throughout the State. Combined meetings, hospital meetings, meetings at our State institutions appeal to the busy doctor as worth while. I am impressed by the character and ability of the officers of local societies. Many of these have served for years with unfailing devotion to duty. If the Massachusetts Medical Society counts for anything in the life of the State it is due, in large part, to these men.

You have read in the JOURNAL the report of the committee on society headquarters. The Massachusetts Medical Society has now, subject to the approval of the Council, a committee room where our members may meet and where our committees may do their work more efficiently. The latchstring is out at 126 Massachusetts Avenue, Room 408.

The ex-airman, whose case I brought to the attention of the Council in October, has been reinstated. He is at Saranac under the care of Dr. Brown. Let us see to it that no worthy invalid soldier suffers neglect through lack of interest on our part.

Our Committee on Uniform Health Examinations reported to the Council in October. This report has met with the approval of our members. What is the next step in this matter? Should not the Society give to its members an opportunity to see the plans of the Committee in use? That is, should we not demonstrate at District meetings health examinations made by our Fellows who have had first-hand experience in this work? These examinations, sufficiently simple to be used in the private office, and yet sufficiently exhaustive to show whether further diagnostic study were necessary, might be made on the Fellows themselves. General health examinations can never be complete diagnostic examinations, but should go far enough into each of the systems of the body to tell whether they are functioning properly or whether they need further time and elaborate study to recognize disease or assure normal health. I would suggest that we select a group of our members, familiar with this work, to go to our Districts to demonstrate their methods, the travel expenses to be met by the Society. Thus may this admirable report of the Committee bear fruit, the growing demand of the public be met, and the cause of sound individualized preventive medicine be advanced.

With the splendid personnel of our Society and with the trust and support given our members by the citizens of the State, let us render a larger service in return.

Dr. Dwight O'Hara reported for the Committee of Arrangements for the annual meeting, Tuesday, June 9, and Wednesday, June 10, in Boston. He said that as there were to be four general meetings of the Society itself it was possible to hold all of them in the audito-

rium of the building of the John Hancock Mutual Life Insurance Company, 100 St. James Avenue, also the Shattuck Lecture. The Cotting Lunch and the annual dinner would be served in the Copley-Plaza Hotel, near at hand to the John Hancock insurance building. He hoped to have interesting moving pictures on medical subjects to follow the Shattuck Lecture, Tuesday evening. As regards the annual dinner at 8 P. M. on Wednesday, he said that his committee would like an expression of opinion from the Council whether to give the dinner free of charge, at an estimated expense of $3500 or more, or to charge $2 a plate to those who attended, at an estimated expense to the Society of $2500. His committee recommended the latter course. On motion, duly seconded, the report was adopted with its recommendations.

Dr. D. N. Blakely read this report of the Committee on Membership and Finance, as to Membership. The report was accepted and its recommendations adopted, by vote:

REPORT OF COMMITTEE ON MEMBERSHIP AND FINANCE, ON MEMBERSHIP

The Committee on Membership and Finance makes the following recommendations as to membership:

1. That the following named nine Fellows be allowed to retire under the provisions of Chapter I, Section 5, of the By-Laws:

1. Abbe, Alanson Joseph, Winter Park, Florida.
2. Drake, William Abram, North Weymouth.
3. Granger, Frank Clark, Randolph.
4. Hoitt, Eugene Gorham, Belmont.
5. Litchfield, William Harvey, Marblehead.
6. Packard, Horace, Boston.
7. Randall, Francis Drew, Tampa, Florida.
8. Tibbetts, James Thomas, Mineola, New York.
9. Townsend, Charles Wendell, Ipswich.

2. That the dues for 1925 of the following named four Fellows be remitted under the provisions of Chapter I, Section 6, of the By-Laws:

1. Ruble, Wells Allen, Watford, Herts, England.
2. Sumner, Harry Herbert, Lowell.
3. Wilder, Edward Wheeler, Madura, South India.
4. Hardwick, Sydney Curtis, Quincy.

And that the unpaid dues up to December 31, 1924, of Bossidy, John Collins, Springfield, be remitted.

3. That the following named fourteen Fellows be allowed to resign under the provisions of Chapter I, Section 7, of the By-Laws:

1. Baxter, Clarence Pennell, San Diego, California.
2. Blair, Orland Rossini, Clark's Summit, Pa.
3. Brennan, John Patrick, Camden, N. J. (with remission of dues).
4. Coolidge, Sumner, Plymouth.
5. Cunning, Daniel Sylvester, New York City (with remission of dues).
6. Fisk, Arthur Lyman, New York City.
7. Hurley, John Joseph, Boston.
8. Kissock, Robert James, New York City (with remission of dues).
9. Leavitt, Frank Clyde, Belmont.
10. MacMichael, Earle Haggett, Englewood, Florida.
11. McCormick, John J., Med. Corps, U. S. Army (with remission of dues).
12. Ripley, Horace Greeley, Brattleboro, Vermont (with remission of dues).
13. Shafer, Rudolph Jonas, King's Park, Long Island, New York.
14. Suarez, Jemaro, Santuce, Porto Rico (with remission of dues).

4. That the following named nineteen Fellows be allowed to change their membership from one District Society to another without change of legal residence, under the provisions of Chapter III, Section 3, of the By-Laws:

One from Bristol North to Plymouth.

1. Stevenson, Willis Mack, North Easton.

One from Middlesex East to Middlesex South.

1. Hodgdon, Ralph Franklin, Winchester.

Seven from Middlesex South to Suffolk.

1. Bailey, Walter Channing, Cambridge.
2. Burnett, Joseph Hamilton, Belmont.
3. Fremont-Smith, Frank, Jr., Cambridge.
4. Lee, Harry Jason, Lexington.
5. Rogers, Albert Edward, Cambridge.
6. Shortell, Joseph Henry, Newton Center.
7. Stein, Louis Charles, Watertown.

Nine from Norfolk to Suffolk.

1. Babcock, Harold Lester, Dedham.
2. Cochrane, Robert Carlyle, Brookline.
3. Ghormley, Ralph Kalb, Brookline.
4. Good, Frederick Leo, Brookline.
5. Howard, Herbert Handy, Brookline.
6. Munro, Donald, Milton.
7. Murphy, William Parry, Jamaica Plain.
8. Overholser, Winfred, Wellesley Hills.
9. Turnbull, John Archibald, Brookline.

One from Norfolk South to Suffolk.

1. Howard, Charles Tilden, Hingham Center.

DAVID N. BLAKELY, *Chairman.*

The Council voted to accept the report of the committee appointed at the last meeting to consider the petition of A. R. Newhall for restoration to the privileges of fellowship, that he be restored under the usual conditions. Petitions to be restored from the following former Fellows were read and committees appointed to consider them respectively:

For W. I. Wiggin:	For W. G. Stickney:
H. W. Jewett	P. P. Johnson
A. R. Gardner	J. A. Shatswell
H. L. Leland	L. C. Swan
For C. A. C. Richardson:	For R. H. Blanchard:
C. E. Mongan	J. B. Thomes
F. E. Bateman	C. T. Leslie
A. H. Blake	Henry Colt

On nomination by the President Dr. C. F. Painter was appointed a delegate to the annual Congress on Medical Education, Medical Licensure, Public Health & Hospitals, to be held under the auspices of the American Medical Association at Chicago, March 9-12, 1925. The President nominated and the Council appointed these delegates to the annual meetings of the New England state medical societies:

MAINE: Ralph W. Jackson, Fall River; Edward Mellus, Newton.

NEW HAMPSHIRE: Edmond F. Cody, New Bedford; Frank R. Ober, Boston.

VERMONT: Frank H. Burnett, Brockton; Eben C. Norton, Norwood.

CONNECTICUT: Walter P. Bowers, Clinton; Frederick E. Jones, Quincy.

RHODE ISLAND: Ransom H. Sartwell, Howard, R. I.; W. O. Hewitt, Attleboro.

Dr. A. K. Stone presented the report of the Treasurer and Auditing Committee and passed around copies of the Reconciliation between the Profit and Loss and Budget for the year 1924, explaining the different items. Voted: To accept the reports. (See Appendix, Nos. 2, 3 and 4, for reports.)

Dr. J. S. Stone read the following report of the committee appointed at the October meeting of the Council as regards headquarters for committees and officers of the Society. It was adopted with its recommendations.

The Committee appointed at the last meeting of the Council "to consider the matter of establishing headquarters for the Massachusetts Medical Society and its officers and committees and for the Editorial Staff of the *Boston Medical and Surgical Journal*" recommend that the Society shall lease Room 408 at 126 Massachusetts Avenue, connecting with the editorial office of the *Boston Medical and Surgical Journal*, at a rental of $388 a year, including janitor service, that equipment secured by the *Journal* be purchased at a cost of $355.55. We recommend that the Society pay the *Journal* $500.00 for the portion of Miss Davies' time which she will give to the work of the Society. We recommend that $100.00 be appropriated for supplies and service, including postage, $25.00 for light and $100.00 for telephone. We ask, therefore, for an appropriation this year of $1468.55, which, after the first year, will probably be about $1100.

We recommend that as far as possible all committees and officers employing clerical help should use the headquarters room and the services of Miss Davies. In this way the expenses of the committees will be decreased, a clerk will always be available, and one person will be familiar with all the details of the work of the various committees and can give any desired information.

As the creation of a medical center at which shall be grouped all health activities has been suggested and as the Boston Medical Library, the *Boston Medical and Surgical Journal*, and the Massachusetts Medical Society would be the chief factors concerned, it is suggested that the Committee keep in touch with these matters and also determine whether there may be closer coöperation between the Society and the Library.

(Signed)

J. S. Stone, *Chairman*,
W. P. Bowers,
T. J. O'Brien,
D. N. Blakely,
S. B. Woodward,
M. V. Pierce,
C. T. Warner,
Henry Colt.

Dr. D. N. Blakely read the report of the Committee on Membership and Finance, as to Finance and it was moved, seconded and carried to accept it and adopt its recommendations. (See Appendix No. 5.)

Dr. C. F. Painter, Chairman of the Committee on Medical Education and Medical Diplomas, made a final report on the undertaking given his committee by the Council last June, namely to investigate, on behalf of the Massachusetts Medical Society, the Middlesex College of Medicine and Surgery and the College of Physicians and Surgeons, Boston, colleges diplomas from which are not accepted from candidates for fellowship. He read letters from Roger S. York, M. D., Dean of the Middlesex College, and from A. S. MacKenzie, Clerk of the Corporation of the College of Physicians and Surgeons, declining to have investigations of their institutions made. On motion, duly seconded, the report was accepted.

Dr. T. J. O'Brien read a report of the Committee on State and National Legislation, and it was accepted. (See Appendix No. 1.)

Dr. Victor Safford made a verbal report for the Committee on Public Health. He said: Your Committee on Public Health will be unable to make a complete report for perhaps a month. During the past calendar year we have continued our efforts to supply speakers for the District Societies and have tried to combat some of the activities of the Medical Liberty League, but our main job has been an effort to investigate the efficacy of methods to control the common communicable diseases, bearing in mind that we are handicapped in our efforts to control such diseases by the fact that in a household the infected person has given the disease to the other members of the household before any methods can be used; and for the reason that to prove the efficacy of methods of control we must take into consideration natural immunity, a thing which we do not understand. We believe that a month from now we shall have something that will be of value to the Society as a whole and to the individual members as well. Moved that the report be accepted; seconded; carried.

On the request of the President Dr. Safford reported as a delegate to the Massachusetts Central Health Council. He said that this Council is a voluntary association made up of two officially designated persons from a number of associations, such as the American Red Cross, American Society for the Control of Cancer, the Massachusetts Dental Society, the boards of health, the Massachusetts Medical Society. He read the constitution of the Council, which he thought sounded a bit ambitious in its aims. The Council meets every two months, has a remarkably full attendance and furnishes an opportunity for an interchange of views among those engaged in state-wide health work; at the last meeting were discussed all the bills affecting matters of public health that are coming up at the present session of the Legislature, so that each member might have an intelligent idea of the motive of each bill. The chair pointed out that Dr. Safford had been instrumental in organizing the Council six or seven years ago; that there are eleven similar state organizations now in existence. Dr. Safford and Dr. Shattuck had represented the Massachusetts Medical Society at the meetings of the Council in the recent past. Dr. Shattuck being away he automatically retires. The President nominated and the Council appointed Victor Safford and J. W. Bartol representatives to the Council for the present year.

On motion by W. B. Jackson it was *Voted*: That the following men be a committee to demonstrate uniform health examinations to meetings of the District Medical Societies:

Roger I. Lee, *Chairman*	Harold M. Frost
F. Dennette Adams	Joseph Garland
Benjamin H. Alton	James Hitchcock
J. Penteado Bill	Frederick S. Hopkins
William B. Breed	Francis H. McCrudden
DeWitt S. Clark	Sylvester F. McKeen
Albert C. England	John H. Taylor

Dr. J. W. Bartol reported for the committee appointed to consider obtaining a bust of Oliver Wendell Holmes for the New York University Hall of Fame, as follows: The committee is of the opinion that the providing of such a memorial is a matter of general concern, and one which does not fall naturally within the purview of this society. They recommend that no action be taken.

(Signed) J. W. Bartol,
Homer Gage,
E. C. Streeter

Dr. A. G. Hurd said he had been much pleased with the annual directory of the Society this year as it appeared in octavo form again. He moved and it was *Voted*: That the Council set its seal of approval on the form in which the annual directory of Fellows has appeared this year.

Dr. J. F. Burnham moved and it was Voted: That the Committee on Medical Education and Medical Diplomas be and they are hereby directed to revise the list of Medical Colleges, diplomas from which are accepted from candidates for fellowship.

Dr. J. F. Burnham spoke as a member of the delegation to the House of Delegates of the American Medical Association, of the matter of periodic health examinations as considered by the House of Delegates last June in committee of the whole. He called attention to the report of the delegation as presented to the Council at its meeting on October 1, 1924, and printed in the Proceedings of the Council. The supplementary report of the Judicial Council of the American Medical Association had been read to the Council at that time, censuring the commercializing of health examinations by stock companies organized solely for profit. He spoke of the high character of the Judicial Council and thought that its findings should have due weight. He offered the following motion: *Moved*, That the Committee on Public Instruction be requested to consider the subject matter of the resolutions adopted by the House of Delegates of the American Medical Association, assembled in committee of the whole, in June, 1924, referring to the supplementary report of the Judicial Council in relation to periodic health examinations by the family physician, and report at the next meeting of the Council; and that this committee confer with the management of the *Boston Medical and Surgical Journal* regarding proper publicity meanwhile.

The motion was seconded by Dr. C. E. Mongan who stated the proposition as "is the medical profession prepared to give its approval to a scheme by which a stock company of laymen is endeavoring to make physical examinations of the community for a certain price?" The life extension institutes are stock companies; they should make a complete exposition of their affairs before they ask encouragement from the profession. He hoped the motion would prevail. On being put to a vote the motion was carried.

Adjourned at 1.22 P. M.

Walter L. Burrage, *Secretary.*

APPENDIX TO PROCEEDINGS OF THE COUNCIL

NO. 1

Report of the Committee on State and National Legislation

It has been said in the past that one of the duties of the President of the Massachusetts Medical Society was to act in an advisory capacity to the members of the Massachusetts Legislature in regard to the solving of medical problems. It may now be said that the efforts of the entire Society are needed to prevent unwise legislation which would either tend to lower our standard, or be detrimental to the public health. Your Committee on State and National Legislation has joined forces with a similar committee representing the Massachusetts Homeopathic Medical Society, as has been the custom for some years, in order that greater influence might be yielded for the common good. The President of the Massachusetts Medical Society is always the Chairman of this Joint Committee and directs its activities. An Auxiliary Committee on State and National Legislation numbering some ninety members is yearly appointed by the President of the Massachusetts Medical Society in order that there may be two or three physicians in each political district. These physicians are instructed in the necessity of stating our views on the important bills to their respective Representatives and Senators, personally. A Representative is naturally influenced by the wishes of his constituents and we lose many votes on important medical measures, at the State House, because physicians at the home town do not bother to call on the local members of the Legislature to discuss the merits and demerits of impending bills.

The policy of your Committee in visiting the various medical meetings throughout the State, especially the combined meetings of District Societies, has borne good fruit, and in every instance where we have asked for assistance from a district president or secretary we have received it willingly and efficiently. We are in closer touch with the dentists, nurses and pharmacists than formerly and are endeavoring to centralize our legislative efforts when possible, as these allied professions are vitally interested in helping us to maintain a high standard of medicine. We are trying to instruct the laymen in medical principles and are doing this through the Friends of Medical Progress, the Medical Directors of Life Insurance Companies, Boards of Health and School Physicians. With our 4100 members scattered throughout the Commonwealth we have unexcelled opportunities for spreading our principles, but we must interest ourselves in this work to accomplish good results. Greater opportunities for efficiency will surely follow the establishing of a headquarters for the various committees at Room 408, 126 Massachusetts Avenue, Boston, adjoining the editorial rooms

of the *Boston Medical and Surgical Journal*. Members of the Society are cordially invited to call personally or by telephone (Back Bay 1887) to get copies of bills or information concerning them.

The bills introduced this year are comparable with those of other sessions.

House 66: An act amending the laws relating to physicians and surgeons. We are recorded in favor of some amendments and opposed to others. Hearing February 2, Public Health. No report has yet been published.

Senate 19: This is the report of the Recess Committee on Registration Laws. The hearing was held before the Committee on State Administration and Public Health, sitting jointly, on February 4, 10.30 A. M. That portion of the report regarding Chiropractors and Midwives will be heard on February 11, 10.30 A. M. and 3 P. M., respectively.

Senate 1: So much as relates to the registration of physicians and others; also so much as relates to Public Health and Mental Diseases. Hearing February 4, 10.30 A. M., Public Health. Dr. Bigelow represented the Society at this hearing.

House 412: Bill to establish in the Department of Education a division for the physically handicapped. Hearing February 5, 10.30 A. M., Room 480. State Administration. Our Committee will not take any action.

Senate 226: Will be heard at 10.30 A. M., February 12. This bill, under petition of J. Oliver Sartwell, is for the registration of Osteopathic practitioners, and regulation of the practice of Osteopathy under a board composed of five graduates of Osteopathic colleges. Our opposition to this bill is based upon our policy of advocating a single standard for the qualification of fitness to practise medicine in this State. The Committee asks for your assistance.

House 366: An act authorizing the Department of Public Health to establish a hospital for the care and treatment of persons suffering with cancer. Hearing February 18, 10.30 A. M., Room 450, Public Health. Your Committee will not take action.

House 973: Bill to provide for the purchase of radium by the Commonwealth to alleviate the suffering and distress caused by cancer. Hearing February 18, 10.30 A. M., Room 450, Public Health. Your committee will not take action.

Senate 171: This bill provides that, in addition to the tenement houses, hospitals should be equipped with a system of automatic sprinklers. Hearing February 19, Public Safety, Room 450, 10 A. M. Your Committee will take no action.

House 365: An act to provide for the inclusion of persons practising Chiropractic in the exemptions now existing under the law. Moved, that any exemptions of Chiropractors be opposed, and we ask for your active coöperation in opposing this bill. Hearing February 25, 10.30, Room 450, Public Health.

House 778: Relative to persons engaging in Chiropractic. Hearing February 25, 10.30, Room 450, Public Health. We voted to oppose this bill.

House 76: An act relative to methods of reporting to the Department of Public Health cases of certain diseases dangerous to the Public Health. Hearing March 4, 10.30, Room 450, Public Health. Your Committee will take no action.

House 972: Resolve to authorize the appointment of a Special Commission for Medical Survey and Research. Hearing March 4, 10.30, Room 450, Public Health. No action.

House 256: Requires all buildings used as hospitals to be of fireproof construction and equipped with automatic sprinklers. Hearing March 5, Public Safety, 11 A. M., Room 450. No action by your Committee.

House 221: Attempted to provide for restricting the sale of patent medicine containing more than 6 per cent. of alcohol. Adverse report accepted by the House.

House 260: Is designed to create a division of preventive medicine under the Department of Public Health, and for a State fund for paying benefits in cases of sickness, accidents or deaths. The Committee on Legislation appeared in opposition because the bill contemplates creating State medicine and is socialistic to an extreme degree. The Committee on Public Health recommends that this be referred to the next annual session. Adverse report accepted by the House.

Senate 108: Petition for an investigation of the affiliations of certain hospital physicians with insurance companies. Adverse report accepted by the House. (The Committee on State and National Legislation found no reason for acting on this bill.) Reported leave to withdraw, Senate.

Senate 169: This is another hardy annual petition of Edna Lawrence Spencer for maternity aid. We intend to oppose it, as usual.

Senate 168: Designed to assist small towns in acquiring resident physicians. No action, as yet, by Committee.

Senate 133: Bill to establish a cancer hospital. As yet, no action by Committee.

Senate 167: To create an investigation relating to prevalence of cancer in this Commonwealth. As yet, no action by Committee.

House 380: An act relative to taxation on sale of intoxicating liquor upon prescription. No action, as yet, by Committee.

Senate 146: An act eliminating the city of Newburyport of the Essex County Tuberculosis District. No action will be taken by your Committee.

House 531: Bill presenting the wishes of the opponents of vaccination who would have exemption from school attendance granted on written statement of parent or guardian who may be opposed to vaccination. Dr. S. B. Woodward has submitted his bill providing that all school children shall be vaccinated, leaving out the specification public as applied to schools, which was in the bill of last year. Your Committee opposes House Bill 531 and actively favors Dr. Woodward's bill. No number has been assigned for the latter bill, but the hearing will be held at the Auditorium on February 17. Federal, State and municipal authorities are interested, and, with the aid of the Society, we hope to pass Dr. Woodward's bill.

Your Committee appreciates the valuable assistance rendered by members of the Society and takes advantage of this opportunity to express it.

THOMAS J. O'BRIEN, *Secretary*.

NO. 2

LETTER OF CERTIFIED PUBLIC ACCOUNTANT

January 27, 1925.

Dr. F. P. Denny, Dr. G. Z. Goodell,
Audit Committee, Massachusetts Medical Society,
Boston, Mass.

Gentlemen:

At the request of your Treasurer, Dr. Arthur K. Stone, we have audited the books and accounts of the Massachusetts Medical Society for the year ended December 31, 1924, and attach:

Schedule A—Statement showing the Assets and Liabilities of the Massachusetts Medical Society, December 31, 1924.

Schedule B—Statement showing the Current Account of the Massachusetts Medical Society for the year ended December 31, 1924.

The cash on deposit in the banks has been reconciled with the bank statement and found to be correct. All known cash receipts have been properly accounted for and disbursements are supported by cancelled checks.

The attached statement of Assets and Liabilities

represents the true condition of the Society to the best of our knowledge and belief.

Respectfully submitted,

HARTSHORN AND WALTER,
Certified Public Accountants.

REPORT OF AUDITING COMMITTEE

We have examined the securities as scheduled in the following account and find them to be as stated.

FRANCIS P. DENNY,
GEORGE Z. GOODELL,
Auditing Committee.

NO. 3

TREASURER'S REPORT

SHOWING THE ASSETS AND LIABILITIES OF THE MASSACHUSETTS MEDICAL SOCIETY
DECEMBER 31, 1924

SCHEDULE A

ASSETS

Cash:			
New England Trust Company		$6,959.49	
Old Colony Trust Company		5,425.87	
			$12,385.36
Investments:			
Shattuck Fund:			
Annuity Policy, Massachusetts Hospital Life Insurance Company		$9,166.87	
Phillips Fund:			
Massachusetts 3½'s Gold Bonds		10,000.00	
Cotting Fund:			
Deposit in Institution for Savings in Roxbury and Its Vicinity	$1,000.00		
Deposit in Provident Institution for Savings in the Town of Boston	1,000.00		
Deposit in Suffolk Savings Bank for Seamen and Others in Boston	1,000.00		
		3,000.00	
Permanent Funds:			
Deposit in Franklin Savings Bank of the City of Boston		1,074.48	
Par value			
$5,200.00 Liberty Bonds 4th Issue 4¼%		5,043.23	
5,000.00 Massachusetts 3½% Bonds 1938		5,000.00	
1,000.00 United States Steel Corporation 5's 1963		1,009.00	
2,000.00 United States Rubber Bonds 5's 1947		1,735.50	
2,000.00 American Sugar Refining Company 6's 1937		1,972.50	
2,000.00 Great Northern Railway Company 5½'s 1952		1,932.50	
2,000.00 Adirondack Power and Light Company 6's 1950		1,970.00	
4,000.00 Public Service Company Northern Illinois 5's 1956		3,640.00	
3,000.00 Dayton Power and Light Company 5's 1941		2,797.50	
3,000.00 Toledo Edison Company 5's 1947		2,805.00	
3,000.00 Cedar Rapids Manufacturing and Power Company 5's 1953		2,805.00	
1,000.00 American Telephone and Telegraph Company 5½'s 1943		985.00	
10,000.00 Winchester Repeating Arms Company Note dated July 7, 1924, due December 31, 1924		9,778.75	
			64,715.33
The Boston Medical and Surgical Journal			1.00
Total			$77,101.69

LIABILITIES

Endowment Funds:		
Shattuck Fund (G. C. Shattuck 1854 Balance 1866)	$9,166.87	
Phillips Fund (Jonathan Phillips 1860)	10,000.00	
Cotting Fund (B. E. Cotting $1,000.00—1876, 1881, 1887)	3,000.00	
		$22,166.87
Surplus:		
Balance, January 1, 1924	49,607.97	
Net Gain for the year ended December 31, 1924—*Schedule B*	5,326.85	
		54,934.82
Total		$77,101.69

STATEMENT
SHOWING THE CURRENT ACCOUNT OF THE MASSACHUSETTS MEDICAL SOCIETY FOR THE YEAR ENDED DECEMBER 31, 1924

SCHEDULE B

CREDIT

Assessments Paid to District Treasurers:			
Barnstable		$233.00	
Berkshire		736.00	
Bristol North		512.00	
Bristol South		1,322.00	
Essex North		1,320.00	
Essex South		1,628.00	
Franklin		281.00	
Hampden		723.00	
Hampshire		1,529.00	
Middlesex East		710.00	
Middlesex North		1,090.00	
Middlesex South		4,412.00	
Norfolk		4,173.00	
Norfolk South		583.00	
Plymouth		729.00	
Suffolk		5,959.00	
Worcester		2,551.00	
Worcester North		642.00	
			$29,133.00
Assessments Paid to Treasurer:		$2,576.06	
Less,—Dues Returned		8.00	
			2,568.06
Total			$31,701.06
Income from Shattuck Fund		$458.34	
Income from Phillips Fund:			
Massachusetts 3½% Bonds		350.00	
Income from Cotting Fund:			
Interest—Institution for Savings in Roxbury and Its Vicinity	$42.50		
Interest—Suffolk Savings Bank for Seamen and Others in Boston	45.00		
Interest—Provident Institution for Savings in the Town of Boston	45.00		
		132.50	
Income from Permanent Funds:			
Interest—Franklin Savings Bank	$48.34		
Liberty Bonds 4¼%	221.00		
Massachusetts Bonds 3½%	175.00		
United States Rubber Bonds	100.00		
United States Steel Bonds	50.00		
American Sugar Bonds	120.00		
Great Northern Railway Bonds	110.00		
Adirondack Light and Power Bonds	120.00		
Cedar Rapids Manufacturing and Power Bonds	150.00		
Dayton Power and Light Company Bonds	75.00		
Toledo & Edison Bonds	150.00		
Public Service Northern Illinois Bonds	200.00		
American Telegraph and Telephone Company Bonds	55.00		
United States Treasury Certificate	212.50		
		1,786.84	
Income from Deposit in Banks:			
New England Trust Company	$265.19		
Old Colony Trust Company	232.49		
		497.68	
Other Income		43.50	
			3,268.86
Total			$34,969.92

DEBIT

General Expense:			
President's expense	$276.83		
Secretary's expense	846.03		
Treasurer's expense	475.52		
District Treasurers' expense	1,759.36		
Censors' expense	481.20		
Delegates' expense	646.30		
Salaries	2,400.00		
Rent	1,337.50		
Miscellaneous expense	51.04		
		$8,273.78	

Boston Medical and Surgical Journal Expense		11,000.00	
Shattuck Lecture		200.00	
Committee Expenses:			
Arrangements	$2,870.92		
Membership and Finance	4.00		
Ethics and Discipline	2.65		
Medical Education and Medical Diplomas	157.25		
State and National Legislation	78.16		
Public Health	60.12		
		3,173.10	
Annual Dividends to District Societies		4,000.00	
Defence of Malpractice Suits		2,669.69	
Cotting Lunches		326.50	
Total Expenses			$29,643.07
Net Gain Transferred to Surplus			$5,326.85

Respectfully submitted,
ARTHUR K. STONE, *Treasurer.*

NO. 4

RECONCILIATION
BETWEEN THE PROFIT AND LOSS AND BUDGET
FOR THE YEAR ENDED DECEMBER 31, 1924

	Profit and Loss Account	Budget Estimate	Difference Under-Estimated	Difference Over-Estimated
REVENUE:				
Assessments	$31,701.06			
Investments	3,268.86			
Total Society Revenue	$34,969.92	$33,000.00	$1,969.92	
Increase in Revenue over Budget		1,969.92		
Total as per Auditor's Report	$34,969.92	$34,969.92		
EXPENSES:				
Salaries of Officers:				
Secretary	$1,500.00	$1,500.00		
Treasurer	500.00	500.00		
Librarian	400.00	400.00		
Officers' Expenses:				
President	276.83	250.00	$26.83	
Secretary	846.03	900.00		$53.97
Treasurer	475.52	500.00		24.48
District Treasurers	1,759.36	1,500.00	259.36	
Censors	481.20	500.00		18.80
Delegates	646.30	850.00		203.70
Rent	1,337.50	1,200.00	137.50	
Journal	11,000.00	16,500.00		5,500.00
Defence of Malpractice Suits	2,669.69	2,000.00	669.69	
Shattuck Lecture	200.00	200.00		
Cotting Lunches	326.50	400.00		73.50
Standing Committees:				
Committee of Arrangements	2,870.92	2,500.00	370.92	
Publications and Scientific Papers		200.00		200.00
Membership and Finance	4.00	25.00		21.00
Ethics and Discipline	2.65	100.00		97.35
Medical Education and Medical Diplomas	157.25	150.00	7.25	
State and National Legislation	78.16	500.00		421.84
Public Health	60.12	600.00		539.88
Dividends to District Societies	4,000.00	4,000.00		
Miscellaneous Expenses	51.04		51.04	
Total Expenses as per Auditor's Report	$29,643.07		$1,522.59	$7,154.52
Total Budget		$35,275.00		
Expenses Over-Estimated		5,631.93	5,631.93	
	$29,643.07	$29,643.07		

Revenue Under-Estimated	$1,969.92
Expenses Over-Estimated	5,631.93
Total Gain over the Budget	$7,601.85
Deduct *Budget Charge to Surplus Account	2,275.00
Balance Transferred to Surplus Account	$5,326.85

*Amount by which Budget Estimated Expenses exceeded Budget Estimated Income.

The Balance transferred to the Surplus Account of $5,326.85 is practically equal to the over-estimated *Journal* Expense, as shown above.

NO. 5

REPORT OF COMMITTEE ON MEMBERSHIP AND FINANCE AS TO FINANCE BUDGET FOR 1925

APPROPRIATIONS

Salaries:		
Secretary	$2,500	
Treasurer	500	
Librarian Emeritus	400	
		$3,400
Expenses of Officers and Delegates:		
President and Vice-President	$250	
Secretary	850	
Treasurer	600	
District Treasurers	1,500	
Censors	500	
Delegates to House of Delegates, A. M. A.	500	
		4,200
Rent		1,200
Boston Medical and Surgical Journal		16,000
Malpractice Defence		2,000
Shattuck Lecture		200
Cotting Lunches		400
Standing Committees:		
Of Arrangements for Annual Meeting	$2,000	
Publications and Scientific Papers	200	
Membership and Finance	25	
Ethics and Discipline	25	
Medical Education and Medical Diplomas (including expenses of Delegate to annual congress at Chicago)	175	
State and National Legislation (including expenses of Delegate to annual congress at Chicago)	500	
Public Health	600	
Public Instruction	300	
		3,825
Dividends to District Societies		4,000
Total		$35,225
Expenses of the Society's headquarters at 126 Massachusetts Avenue (provided the Council accepts the report of the Committee appointed at the last meeting and adopts its recommendations)		1,475
Total		$36,700
Income as Estimated by Treasurer		35,000
To be taken from Surplus		$1,700

DAVID N. BLAKELY, *Chairman.*

The Drive of Civilization, Fatigue, Disease, and Decadence

No priestess on a tripod is needed to warn us that the highly civilized races of mankind are going backward physically; that the advanced races of the world are, as Wiggam says, biologcially plunging downward. Wiggam, in his New Decalogue of Science, reminds us that civilization always destroys the man who builds it; that many diseases are chiefly the by-product of our civilization; that the tension diseases—cancer, goitre, heart disease, degenerative diseases of the arteries, focal infections in teeth due to improper diet, neuroses, psychoses, insanity and multiform minor mental and nervous derangements, which affect men's behavior unfavorably, are increasing.—*Excerpt from Address of Julius Rilus Eastman read at Meeting of Michigan State Medical Association.*

Case Records
of the
Massachusetts General Hospital

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICO-PATHOLOGICAL EXERCISES

EDITED BY
RICHARD C. CABOT, M.D., AND HUGH CABOT, M.D.
F. M. PAINTER, A.B., ASSISTANT EDITOR

CASE 11091

MEDICAL DEPARTMENT

At his first admission the history and examination of this patient, an American shopkeeper of forty-five, were negative except for hemorrhoids and the heart. The apex impulse was one and a half inches outside the nipple line. There was a loud whistling systolic murmur transmitted to the axilla. The hemorrhoids were cauterized. A year later he reported a perfect result.

Eight months later he reëntered. He now gave a history of chancres two years earlier. For a week he had not felt well. For six days he had had cough. Then he had severe pain in the back of the left thigh and calf running up into the small of the back. Two days before admission this became so severe that he had to go to bed. He had also had severe pain in the left knee-joint, and of late had had some abdominal pain radiating to the back.

Examination showed a barrel-shaped chest. The apex impulse of the heart was in the sixth space just outside the nipple line. There was no enlargement to percussion. The action was regular, the sounds of good quality, the second sound at the apex ringing. A systolic murmur at the apex replaced the first sound and was transmitted to the axilla. The aortic second sound was greater than the pulmonic second. The pulses were normal, the arteries palpable. The lungs were hyperresonant. The breath sounds were loud. Expiration was prolonged. There were occasional moist râles. There was considerable general tenderness in the abdomen. The genitals were normal. The left knee-joint contained an excess of fluid, the patella floating. There was great tenderness about the joint. The left leg was everted. There was much tenderness over the course of the left sciatic nerve and calf. The pupils and reflexes were normal except that the left knee-jerk was not obtained.

The temperature at entrance May 6 was 102.5°, after May 9 normal. The pulse was 108-60. The respirations were normal. The urine was normal in amount. The specific gravity ranged from 1.010 to 1.023. There was the slightest possible trace of albumin at both of two examinations. The hemoglobin was seventy-five per cent., the leucocytes 6,600.

Under salicylates the pain and swelling of the knee-joint disappeared. May 15 the patient was discharged "well."

He was admitted for the third time January 8, five years later. For the past few years he had urinated four or five times at night. For two months he had had considerable cough which disturbed his sleep, loss of appetite, dry and slightly sore throat, and occasional vomiting. He had lost some weight and strength recently. January 6 he had soreness at the base of his neck, all over the chest, and along the right costal margin, worse on cough or deep inspiration. For a day or two he had been raising bloody sputum.

Examination showed a well nourished, full-blooded man with frequent paroxysms of violent coughing, considerable dyspnea, and slight periodic audible respiratory wheeze. The skin was somewhat pale. The teeth were a few decayed snags, with pyorrhea. The apex impulse of the heart was in the fifth space fifteen centimeters from the midsternal line, five centimeters outside the nipple line, corresponding with the left border of dullness. The right border of dullness was four centimeters from midsternum. A systolic scratchy murmur was heard at the apex. The pulmonic second sound was accentuated. The pulses were normal, the artery walls moderately thickened. The systolic blood pressure was 130. Expiration was prolonged throughout and accompanied by many wheezes, especially at the apices in front. There were a few inspiratory crackles. The liver dullness extended from the sixth rib to two centimeters below the costal margin; the edge was not definitely felt. The urinary output was normal. The urine was light brown to black-brown at the first seven of sixteen examinations, cloudy or smoky at all the rest, high colored at three. The specific gravity was 1.009 to 1.013. There was albumin at all but the last examination, a large trace at the first six, a trace to a very slight trace later. The sediment showed blood decreasing from much to rare red cells, then none; at the end many red corpuscles to much free blood. The first eight examinations showed many brown granular and blood casts and hyalin casts with cells and blood attached. Later there were few, and the blood disappeared. At entrance the hemoglobin was 80 per cent., the leucocyte count 34,200; the reds showed slight achromia and numerous large polychromatophilic cells. Later the leucocyte count fell, reaching normal January 17.

During the first week in the hospital the patient ran a temperature of 99.7° to 104.2°; the pulse was 71-100, the respirations 24-48. During the next week the temperature was 98.2°-101°, the pulse 64-95, the respirations 18-37. The third week the temperature gradually reached normal and the pulse and respirations were normal throughout. During the first three days the

patient had violent paroxysms of coughing, raising considerable thick tenacious brown sputum showing some fresh blood and considerable pus; no tubercle bacilli. He had slight nosebleed much of the time from entrance. A large number of bloody crusts formed on the nose and upper lip. He bled easily also from the ear. A purpuric spot was left after the injection of morphia. After January 11 the cough was much less severe. January 14 there was marked pain on inspiration and a friction rub in the left axilla. He made good general improvement. The tendency to bleed diminished. The urine showed much less color than at first. January 18 he had a small bloody stool. January 22 there was a diastolic blow transmitted down the left border of the sternum and a slightly collapsing pulse. The apex impulse was localized and forcible. The patient said he felt strong and well. He continued to improve rapidly. January 29 his lungs were clear. That day he was discharged.

After leaving the hospital he felt fairly well, though he had severe paroxysms of cough. He was able to do a little work by resting every few minutes to ease his dyspnea. When walking he had to rest every few steps. His bowels were constipated. He slept poorly because of attacks of coughing. There was no sputum. He had much dizziness. In the late spring his feet and ankles began to swell at night and he had severe cramps in his legs. He urinated three times at night. By May 9 the cough had become almost incessant and dull abdominal pain had developed.

At his fourth admission to the hospital, May 12, five months after his last discharge, he was well nourished, but looked old. He sat propped up in bed breathing very rapidly, with frequent attacks of severe cough during which he became cyanotic and after which he gasped for air. The skin was pale and moist. Over the arms and in great numbers on the legs were purpuric areas. The mucous membranes and throat were pale and dry, the throat and pharynx crusted. The tongue was dry, furrowed and cracked, with a brown crust. The teeth were a few carious stumps. There was marked pyorrhea. Glands the size of peas were felt in the neck and axillae and the size of beans in the groins. The apex impulse of the heart was seen and felt in the fifth space fourteen centimeters from midsternum and six and a half centimeters outside the nipple line, corresponding with the left border of dullness. The right border was four and a half centimeters to the right. The action was regular, not rapid. The sounds were clear and of good quality. At the apex was a loud harsh systolic murmur, heard also over the precordia and transmitted to the axilla and back. The pulmonic second sound was slightly accentuated. There was slight lateral excursion of the brachials. The systolic blood pressure was 105. The lungs were rather hyperresonant except at both bases posteriorly, where there was slight dullness. The expiratory murmur was rather prolonged. A few squeaks were heard throughout, and below the angles of the scapulae numerous fine moist râles. The abdomen was held rigid. The liver dullness extended from the sixth rib to the costal margin. A tender edge was felt indistinctly at the level of the umbilicus in the nipple line. Both shins and ankles showed considerable soft edema.

During his eleven days in the hospital the patient ran an irregular temperature from 97.9°, to 101.6° with two intervals of normal temperature for two days and a terminal rise to 103.7°. The pulse ranged from 85 to 110, with a terminal rise to 135. The respirations were 24 to 43. The output of urine was normal except on May 14, when it was ℥ 73. The urine was smoky or cloudy at all of five examinations, bloody at two, high colored at the other three. The specific gravity was fixed at 1.011-1.012. A trace to the slightest possible trace of albumin and a few to rare red blood corpuscles were found at all examinations. At one a guaiac test on the supernatant fluid was strongly positive. Hyalin, granular and cellular casts were found at all examinations, with blood cells attached at the first two. The hemoglobin was 50 to 60 per cent., the reds 3,000,000, showing achromia at one of three examinations, macrocytosis at two, variation in size at one, in shape at another. The leucocytes and differential count were normal until the day of death.

The patient seemed at times rather childish. He had sudden attacks of severe dyspnea which usually could be easily quieted without medicine, though at times there was very marked genuine dyspnea requiring morphia. This grew much worse. He had several very severe nosebleeds. The gums and lips bled slightly at times. May 19 an ulceration on the nasal septum was touched with chromic acid. May 21 a localized patch of large vesicles developed on the upper right eyelid. The stool was hard costive pellets imbedded in a little fresh blood. May 22 the sputum was a membranous looking material showing a few Gram-positive diplococci. The patient was unable to swallow. A throat culture was taken. The leucocyte count was 14,000. The heart was very irregular, the pulse weak. May 22 he suddenly collapsed and died.

Discussion

by dr. richard c. cabot

notes on the history

1. This is rather a good example of how much good percussion is. If we can feel a heart we know we have it. Here we felt in the sixth space outside the nipple line, and yet percussion does not show any enlargement.

2. The "second sound at apex" is of course the aortic second, and therefore makes us think we are going to hear the same thing when we come up into the second right interspace.

3. This is just exactly the type of chest examination as a result of which we used to say "emphysema." We carefully do not say so today.

4. We cannot get a knee-jerk when the patient is in so much pain,—we do not try practically.

5. The sort of soreness in the chest which he had at the third admission often comes from the cough itself. The muscles are strained. I do not see very well what else it could be.

6. A "full-blooded" means a "red-faced" man.

7. The urine gives the picture of acute nephritis unless there is a definite local lesion in the bladder or elsewhere in the urinary tract to account for the blood. As the blood is attached to casts, which could not have come about at all from local lesions, I should suppose this was an acute nephritis. Acute nephritis can do all that.

8. But we have to have something more than acute nephritis to cause all this fever. This is a striking chart, such as we should get with pneumonia, and I rather think he had it. We have no signs, but bronchopneumonia does not necessarily give any signs.

9. The crusts on the upper lip probably mean herpes, which would go with pneumonia.

The lungs were clear about three weeks after he came in.

I should say the diagnosis was pneumonia and acute nephritis at this time, with a tendency to bleed which we do not quite understand, possibly from an acute endocarditis which perhaps is giving him his diastolic murmur. We can tell better next time, when we see what the heart shows.

What could give him this dyspnea after leaving the hospital? As far as I see, only his heart, unless he has an emphysema, which I discount.

NOTES ON THE PHYSICAL EXAMINATION

I should suppose these glands were perfectly normal.

Again we have the lungs of what we used to call emphysema.

This is the picture of a failing heart without valve lesions and probably an arteriosclerosis, but without hypertension so far as we know. We do not yet know about his urine. The kidney of course could have been the source of it.

A PHYSICIAN: Could that constant abdominal pain have been due to coronary occlusion?

DR. CABOT: No; it would have to be sharper and accompanied by more failure of the pulse. There is a good pulse and nothing else to show that a vessel has been suddenly blocked.

A PHYSICIAN: Would it indicate infarcts?

DR. CABOT: No, I should not say so. We can diagnose infarcts only by sudden and sharp pain, not by a dull pain. I should say that the pain was due to a congested liver. We know it is congested. We have felt it down near the umbilicus. And that is enough to give this pain.

The old trouble he has had with bleeding comes back several times.

The patch on the eyelid was herpes, I suppose, in rather an unusual place but not impossible.

DIFFERENTIAL DIAGNOSIS

Let us start with the things we know relatively well. His heart is big, and his heart has shown no constant evidences of a valve lesion, I mean of a chronic endocarditis. So I should say we should expect a hypertrophied and dilated heart of the type that we usually see associated with hypertension, sometimes without hypertension. I should say he would probably have some arteriosclerosis, but he certainly ought to have a chronic nephritis. As to the type I have nothing to say. I cannot any longer predict types at all—chronic nephritis is all I can say.

Should his lung show anything? I do not think his lungs played any important part in his death. If he had emphysema we do not know how to recognize it. I should say that he did not have it. He may have had a bronchopneumonia. We cannot say. It may have been a reason for the temperature. There may be infarcts in his lungs—I should be pretty sure in his lungs and elsewhere, it has been going on so long.

What is the cause of his bleeding? Acute endocarditis naturally comes to mind, and I do not know any way to exclude it. He has had a murmur, he has had fever, and these purpuric spots. I do not see any way to exclude it. We cannot say more unless there were a diastolic murmur coming in or some sudden change in a murmur such as we suppose accompanies a change in the vegetations.

What can be the cause for the purpura? Chronic nephritis itself is enough, as we all know, for "renal nosebleeds," so-called, that is for bleeding from a sound kidney and also for bleeding elsewhere. So we do not need to find any other cause for that bleeding than the kidney trouble which I confidently expect will be shown.

We have no evidence of prostatic trouble or tumor, or hydronephrosis. We have no reason to suspect any other organ in the body than those I have named, so far as I see.

A PHYSICIAN: Would you expect adhesive pericarditis in this case?

DR. CABOT: I do not see why. It is a diagnosis that nobody can ever exclude, but I do not see why we should look for it. The absence of murmurs would not make me favor pericar-

ditis. Pericarditis often produces endocardial murmurs. All we know today about pericarditis is that we cannot recognize it. We may suspect it in young rheumatic subjects with tremendous hearts and ascites; with old people we often find it without a single thing in life to put us on the track. So I should say in answer to your question that it may perfectly well be there, but you have no reason to say so and neither have I.

BACTERIOLOGICAL REPORT

A throat culture reported after death was positive for Klebs-Loeffler bacilli.

CLINICAL DIAGNOSIS

Chronic bronchitis and emphysema.
Mitral regurgitation.
Chronic passive congestion, general.
Purpura.
Hematuria.
Secondary anemia.
Diphtheria.

DR. RICHARD C. CABOT'S DIAGNOSIS

Chronic nephritis.
Hypertrophied and dilated heart.
Acute endocarditis.
Chronic passive congestion.
Diphtheria.

ANATOMICAL DIAGNOSIS

Chronic fibrous and acute vegetative endocarditis of the aortic and mitral valves.
Hypertrophy and dilatation of the heart.
Chronic passive congestion of the lungs and liver.
Arteriosclerosis.
Lobar pneumonia of the upper lobe of the left lung.
Soft spleen.
Acute degeneration of the liver.
Secondary anemia.
Arteriosclerotic degeneration of the kidneys.
Streptococcus septicemia.

DR. RICHARDSON: The head was not examined. There is nothing about diphtheria in our record here. Scattered over the lungs were numerous irregular discrete and confluent purpuric-like areas. The culture taken from the heart blood yielded a good growth of the streptococcus. There was a slight amount of pale fluid in each pleural cavity and a few adhesions. The right lung showed chronic passive congestion, no pneumonia. The lower two-thirds of the upper lobe of the left lung showed frank pneumonia with the usual pleuritis.

DR. CABOT: No emphysema?

DR. RICHARDSON: No.

DR. CABOT: You would have put it down if it had been there, wouldn't you? It is an obvious thing?

DR. RICHARDSON: Yes, it is obvious.

Scattered along the aorta there was a slight to moderate amount of fibrous sclerosis.

The pericardium contained a slight amount of pale fluid. The heart weighed 565 grams,—considerably enlarged. The myocardium was thick. The mitral valve measured 9.5 cm., the aortic 9 cm. The tricuspid and pulmonary valves were negative. Scattered along the curtain of the mitral valve were numerous fibrous areas and nodules, causing some deformity, and planted on these were smaller and larger masses of soft frank spongy vegetations,—acute endocarditis on a basis of chronic.

The coronaries were free, capacious, and showed only a slight amount of fibrous sclerosis.

The liver and the other organs showed chronic passive congestion.

There was some question as to anemia in this case. It was considered at that time that there was some anemia, but it was secondary and not primary. The kidneys weighed 345 grams. From the description there was nothing but arteriosclerotic degeneration, that is, arteriosclerosis of the vessels and some of the capillaries of the glomeruli and scattered foci. At that particular time it was not regarded as sufficient to call it arteriosclerotic nephritis.

DR. CABOT: Do you suppose you have a slide upstairs?

DR. RICHARDSON: Yes. But it is described here.

DR. CABOT: I should like to have it looked over some time. We certainly were flat-footed in the diagnosis.*

A PHYSICIAN: What about the Klebs-Loeffler?

DR. CABOT: It was a terminal infection. I have no doubt it contributed to the death. But you see there is plenty more infection, with the acute endocarditis throwing emboli around. I suppose it perfectly might have been in the upper air passages, and if you had not been told about it you would not look.

DR. RICHARDSON: No; but a case of Klebs-Loeffler would be taken care of in a proper fashion.

DR. CABOT: One would think they would have made more of a fuss about it. The chief mystery to me is that Dr. Richardson so far refuses to back us up on the diagnosis of nephritis.

CASE 11092

MEDICAL DEPARTMENT

An Irish housewife of fifty-eight came to the Emergency Ward November 13.

F. H. Her husband had been an invalid for two years following an operation for an abscess of the hip.

*On reëxamination of the slide Dr. Richardson confirmed the previous finding

P. H. She had measles and whooping cough in childhood. She had had three miscarriages. For years she had had poor vision. She had frequent dry cough. She usually was dyspneic upon exertion. For several years she had frequent attacks of severe colicky abdominal pain and vomiting, often centering over the umbilicus, where she had a large hernia, and ceasing after operation upon this ten years ago. For years she had urinated three or four times at night. For years she had had varicose veins. Three years ago those on the left leg broke down, but healed after she stayed in bed. At intervals since that time the leg had given her considerable pain. The right leg had never given any more than slight discomfort until the present illness. A month ago sores appeared on the right leg.

P. I. November 10 she was seized with chills alternating with hot flushes, and the right foot, which had given some discomfort at intervals for months, was acutely painful, particularly the great toe and the ball of the foot. The chills ceased, giving place to a generalized continuous burning febrile sensation. The next morning she seemed disoriented for an hour or two and did not recognize relatives. The mental condition cleared up later. The right foot continued to pain and the leg began to be tender above the ankle. The following day the pain and fever continued and the tenderness extended higher on the leg. For two days she had had considerable nausea but no vomiting. At admission her condition was practically the same except that the tenderness had mounted still higher. She had been in bed for three days.

P. E. An extremely obese woman, incontinent of urine and feces, complaining of nausea, burning and pain in the right foot and leg. External strabismus. Sclerae faintly jaundiced. Teeth poor. Pyorrhea. Apex impulse of the *heart* not found. Measurements not recorded. Left border of dullness indistinct but seemed not to be abnormal. Sounds distant. Action regular. No murmurs. *B. P.* 80/50. *Lungs* clear in front. A few moist crackles just below the angles of the scapulae. *Abdomen* very large and flaccid. A long transverse scar just below the umbilicus near the center of which was a fluctuating round swelling, tympanitic, not tender, the size of half an orange. It could not be entirely reduced; on pressure gas could be felt to gurgle out of it. *Extremities.* The skin over both lower legs anteriorly was bluish-red, thick, dry and cracked, that over the feet scaly and dry. Right leg somewhat swollen, redder than left, and very tender all over. Over the tibia just above the ankle were two small areas recently broken down, now covered with scabs. Just above the knee a distinct irregular red border could be seen. *Pupils.* Right greater than left. Both normal in shape and reactions. *Reflexes.* Right knee-jerk not tried. No clonus or Babinski.

T. 100°-103.2°, with afternon rise on four of the nine days. *P.* 96-140. *R.* 20-43. *Urine.* ℥ 30 on the one occasion recorded, sp. gr. 1.030-1.012, high colored at one of three examinations, a very slight trace of albumin at another, occasional leucocytes at one (no catheter specimen). *Blood* not recorded except leucocytes 28,600-13,300. *Wassermann* negative.

The day after admission the patient looked better, breathed more easily and coughed less. She continued to be incontinent, complained a little of abdominal cramp, and had some nausea. The red area on the leg had extended about an inch and a half higher. The temperature was a little lower. A blood culture showed Gram-positive spore-bearing bacilli in one flask, no growth in the other. November 16 the leg looked slightly better, though she complained of pain in it. The process was extending only very slowly. Elevation did not seem to influence its course. The color faded perceptibly. She complained, however, more than ever of pain. November 20 the leg looked on the whole better. The color was fading in the region about the knee. There was fair circulation in it and in the foot. The tenderness was decreasing and she complained of less pain. She vomited much less than the day before. The bowels moved normally. The temperature remained about the same. She coughed. Early the morning of November 22 she died.

Discussion

by dr. maurice fremont-smith

notes on the history

There is little in the past history. The three miscarriages must be remembered as a possible indication of lues. The poor vision we do not know enough about to lead us in any direction. Her dyspnea on exercise may or may not be due to failing heart. A woman who has varicose veins of both legs is apt to be obese and her dyspnea on exertion might be secondary to that. The fact that her varicose ulcer healed after she stayed in bed would suggest that it was not luetic, although of course we do not know what medication she may have had.

In other words, she developed some type of acute infection with marked toxemia and a progressive nature.

notes on the physical examination

We do not know which eye showed strabismus. We do not know that she had not always had strabismus. If it were of recent onset it would be an important finding.

Miss Painter: "Right" has been written and has been carefully erased.

Dr. Fremont-Smith: The jaundice is another sign of toxic absorption.

Of course it is important to know whether or not this is a big heart, and we have nothing here to help us. This is a fat woman. We cannot feel the apex and we cannot make out the left border. We have not a very high blood pressure to make us feel certain that the heart must be definitely enlarged. I feel that if they had looked for the point where the apex impulse was heard loudest they might have determined with some degree of certainty the size of the heart.

DR. CABOT: I think that is true if the sounds are anywhere near the ordinary intensity. This record says that the sounds were distant, and where they are so poor I doubt whether it is possible to use them as localizing signs.

DR. FREMONT-SMITH: Her blood pressure of course is extremely low and would suggest shock or a very markedly failing myocardium,—hardly that however without more signs of actual congestive failure.

There is nothing very definite in the lungs.

She has not had her ventral hernia entirely repaired, but there is no evidence that she is in any trouble from that.

I do not see any reference here to the lymphatic glands, which are too important to be left out, although they probably will show nothing.

MISS PAINTER: They were normal.

DR. FREMONT-SMITH: The fact that one of the pupils is greater than the other makes us think of some central nervous pathology. On the other hand if she has had strabismus for some time with partial blindness of one eye she may have had one pupil larger than the other without any involvement of the central nervous system.

She has of course a spreading infection of the leg, and we have here a suggestion that it may have been erysipelas. The cardinal sign of erysipelas is the raised, distinct reddish border. On the other hand erysipelas does not usually cause so much pain as this. Perhaps we would better go further before we try to make a differential diagnosis.

I think we can say on the basis of this examination that the kidneys are normal.

Probably we may throw out these blood cultures. One was negative, the other showed an organism which we can hardly conceive as being related to this condition.

Of course we might think of anthrax. The whole picture is not that of anthrax, which occurs first as a small blister surrounded by a purplish areola; the bleb becomes dark, then necrotic, with a bluish induration around it. This is a much more localized affair with very much less generalized symptoms and lower temperature. In anthrax the blood culture is very rarely positive.

DIFFERENTIAL DIAGNOSIS

There are two things to discuss: in the first place, what the condition was on her leg, and in the second place, why when she apparently was getting better she died. We have to consider erysipelas. We have to consider a deep cellulitis. We have to think of phlebitis.

Erysipelas usually has less pain, and the temperature when it goes up stays up five or six or ten days. It is rare in erysipelas to have this remission of the temperature. (In the chart the remission is not so striking as it seems from the record.) The other possibility is that she was infected through the small ulcers that appeared about a month ago on the lower leg, and developed a cellulitis of the foot extending up the leg, probably streptococcal in origin. Of course the pathology in the two conditions, cellulitis and erysipelas, differs not very greatly. In one case the superficial structures, the skin and subcutaneous area, only are involved, in cellulitis the deeper structures. Both conditions can give rise to thrombus formation, and I think we have to assume a thrombosis and infarction to explain her death.

Of course phlebitis would even more easily explain the fact that she may have had an embolus. However, this is hardly the picture of primary phlebitis, where one usually finds a single vein—more frequently, in the leg, the vein in the calf —which is extremely tender and hard, over which there is localized redness, hardly ever a diffuse cellulitis such as this evidently was. Of course such a cellulitis or erysipelas might cause phlebitis and thrombus.

I do not see how we can say anything about her heart. Her kidneys were probably normal. I doubt if we shall find anything in her lungs.

A PHYSICIAN: Do we need to consider any deep infection such as osteomyelitis?

DR. FREMONT-SMITH: I think we should consider osteomyelitis. It is apt, however, to be pretty well localized to the bone in which it begins. If she had had an osteomyelitis of the great toe and the ball of the foot it would be more likely to find the signs localized to this region.

DR. CABOT: I do not remember having seen an acute osteomyelitis at this age. I should lay a little more stress than you do on the possibility of bronchopneumonia. That would account for her death, and she did have a cough. That would account for her death without supposing any thrombus or embolism, which we do not like to assume when we have apparently nothing sudden.

DR. FREMONT-SMITH: Of course we have no description of her death, and the feeling has been that they were expecting her to get well.

A PHYSICIAN: Is there a possibility of gas bacillus infection?

DR. FREMONT-SMITH: It would have been a much quicker thing. She would have blown up very much quicker. They would have got crepitus around the inflammatory area.

MISS PAINTER: The last words in the record are, "The contributing cause is evidently pulmonary."

DR. CABOT: So they knew more than they told us. Perhaps they were wrong, but that is what they thought.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Streptococcus cellulitis, right leg, non-traumatic.

DR. MAURICE FREMONT-SMITH'S DIAGNOSIS

Streptococcus cellulitis.
Thrombosis.

ANATOMICAL DIAGNOSIS

1. *Primary fatal lesion*

Cellulitis of right leg.

2. *Secondary or terminal lesions*

Septicemia, streptococcus.
General peritonitis.
Cirrhosis of the liver.
Hypertrophy of the spleen.
Cholelithiasis.
Hemorrhagic edema of the lungs.
Hypertrophy and dilatation of the heart.

3. *Historical landmarks*

Scar of old operation wound of the abdomen.
Abdominal intestinal hernia.
Ulceration of small intestine.

DR. RICHARDSON: There was the long scar mentioned, extending from side to side in the anterior abdominal wall, and in the region of the scar, peritoneal side, a couple of coils of small intestine were incarcerated. They were constricted somewhat but still were free and their walls intact.

The peritoneal cavity contained a small amount of thin purulent fluid, general peritonitis.

DR. CABOT: But that did not come from the intestine, from this hernia?

DR. RICHARDSON: No. About the liver there were vascular adhesions. The liver was large and showed frank cirrhosis. The gastro-intestinal tract was otherwise negative except for a few superficial small ulcers of the small intestine, not tuberculous.

The lungs showed a moderate amount of hemorrhagic edema.

The heart weighed 475 grams, was moderately enlarged, with thick myocardium, slight dilatation, negative valves. The aorta showed a slight amount of fibrous sclerosis and a slight amount of fibrous sclerosis was present in the great branches, but all told, negative. The gall-bladder contained twenty stones, five mm. to seven cm. in diameter. The mucosa was negative. The bile-ducts were free and negative. The spleen weighed 1110 grams, greatly enlarged.

DR. CABOT: Was it down at all below the ribs?

DR. RICHARDSON: Yes.

DR. CABOT: So we might have felt it during life.

DR. RICHARDSON: She was a very stout woman.

DR. CABOT: Then if she had not been so stout we might have felt it.

DR. RICHARDSON: The tissue of the spleen was plump, brown-red and a little mushy in places, presumably congestion in association with the cirrhosis of the liver. On the right leg there was an area of loss of substance and purulent infiltration of the subcutaneous tissues, an area of infection and cellulitis.

Culture from the spleen showed streptococci.

CASE 11093

SURGICAL DEPARTMENT

An Irish housemaid of forty-eight entered August 21. She gave a history of measles, mumps and scarlet fever in childhood. Otherwise she had always been strong and well except for dyspepsia of several years' standing. For the past two years her catamenia had been very irregular and scanty. The day before admission, three hours after eating cabbage, she was seized with severe abdominal cramps, nausea, vomiting and diarrhea. The symptoms continued all through the day of admission, and she had vomited and retched continually. The vomitus was dark colored and watery. She could not sleep. The cramps were relieved by pressure.

Examination showed a slightly obese woman with markedly irregular heart action and pulse. The heart was not enlarged and showed no other abnormalities. The base of the right lung in front and behind showed slight dullness, many coarse moist râles, and diminished tactile and vocal fremitus and breathing. At the left apex behind there was slight dullness with marked bronchial breathing. The abdomen was much distended, the navel flushed. The muscles were held rigid. There was slight fullness in the epigastrium and slight general tenderness. To the right of the umbilicus was a soft tumor about an inch in diameter under the skin. About three inches below this was a small hard subcutaneous nodule, and in the epigastrium a similar one.

The temperature was normal, the pulse 128 to 131, the respirations 23 to 36. The urine showed a very slight trace of albumin; otherwise it was not remarkable. The hemoglobin was 70%, the leucocytes 27,400. The vomitus was dark brown. Microscopical examination showed it to be made up of fibrin and normal red blood corpuscles.

The night of admission the patient did not seem in poor condition. The pain was easily relieved by a hot water bag. After midnight she vomited a few times, castor oil and some food

and bile. In the morning she vomited black material which proved to be mostly blood. Her abdomen was much distended and her face ashen. She continued to vomit quarts of black material. Operation was done that morning. The patient died on the operating table.

Discussion

by dr. edward l. young, jr.

I think it perhaps will be interesting to compare whether or not surgical histories have developed since this record was written. Dr. Cabot still likes to designate a good many as "surgical histories" as contrasted with "medical,' and as I look at this it looks very "surgical."

Miss Painter: It was in fact medical.

Dr. Young: The account of the catamenia is consistent with beginning menopause.

"The cramps were relieved by pressure." That is an interesting observation. I wonder if it was brought out by a leading question or was volunteered.

Dr. Cabot: Isn't that often so?

Dr. Young: If they are cramps it is. When it is an infectious thing it is not.

Of course this story is consistent with acute indigestion. I did not believe up to a few years ago that any medical condition could cause really severe abdominal pain, but acute indigestion can. On the other hand with such a story as this we must seriously consider the acute emergencies, particularly the question of a ruptured ulcer. She has had dyspepsia for several years. I wish they had got a little more definite story about what that dyspepsia was, although of course we know that rupture can be the first sign of a peptic ulcer. We have to consider all the emergencies. Even an acute appendix may do this sort of thing, although we do not expect diarrhea with that. Pancreatitis may be present, because that also comes on and is very often preceded by the dyspepsia of biliary tract disease. Or an acute cholecystitis might cause this.

The one thing in certain of these cases that will differentiate is the feel of the abdomen, because a perforation results in the most board-like abdominal feel of any surgical emergency that we have, and at first, within the first twenty-four hours always, often within the first forty-eight hours, the temperature is normal because the perforation coming in the duodenum or near the pylorus is practically in a sterile area. They say that the muscles were rigid. That of course rules out such a condition, for instance, as a sudden acute intestinal obstruction, because in that condition the abdominal muscles are not rigid. In perforation, however, the muscles are rigid. So that we have to assume a very considerable insult to the peritoneum.

Could it be a pancreatitis? That cannot be ruled out. An acute appendix would have a temperature presumably, and of course the perforation would have leucocytosis such as this has, just the same as the other conditions.

I do not see how we have any right to say anything more than perforation as the first diagnosis, and on that basis the only thing that will do any good is immediate operation. It has been going on twenty-four hours anyway.

A Physician: What is the significance of the blood in the stomach?

Dr. Young: That can of course go with perforation. The continued vomiting is the vomiting that might go with the obstruction.

I do not quite understand why they did not operate at once, because it says, "The night of admission the patient did not seem in poor condition." She is having some bleeding into the intestinal tract, and the best bet is that we are dealing with an ulcer which has perforated and is also bleeding. I should think if they had had the courage of their convictions and not operated until this stage of the game they might have let her die in peace, because it seems that with the story we have the only thing to do was to have given her the chance when she first came. This is a short story, but it seems a pretty definite one. I do not see how they could have seen more in the patient than we can gather.

I should put the diagnosis, first, perforated ulcer; second, and a very poor second, any one of the other acute surgical emergencies such as pancreatitis or appendicitis. These tumors they speak of I assume are not in the abdomen but simply lipomas or fibromas in the abdominal wall.

Dr. Cabot: You do not think of appendicitis seriously?

Dr. Young: Not seriously. We have always to bear that in mind, because it can always do any one of these things. I saw a case here one night last winter, with the most severe symptoms such as this, with a board-like abdomen. There was no question in the mind of any of us but that she had a perforation. She had an appendix.

dr. young's pre-operative diagnosis

Ruptured peptic ulcer.

operation

No pre-operative diagnosis is recorded. Gas and ether. Incision made in the median line from umbilicus to pubes. Considerable blood tinged fluid escaped when the peritoneum was opened. A portion of the small intestine about twenty-four inches in length seemed to be gangrenous and distended, apparently at the beginning of the ileum. The demarcation line was distinct. The mesentery was thrombosed and friable.

Further Discussion

A mesenteric thrombosis, not a perforation. That is all we are told. My differential was made

on the fact that the abdominal muscles were held rigid. I do not see that there is anything more to say.

twist in the jejunum in the region of the involved portion."

DR. YOUNG: A volvulus.

Photograph by Mr. Lewis S. Brown.
DR. OSCAR RICHARDSON.

DR. RICHARDSON: There is a note here that I think Dr. Young should know. Apparently Dr. Scudder must have told me himself, because I have recorded, "The surgeon, Dr. Scudder, states that at the time of operation there was a

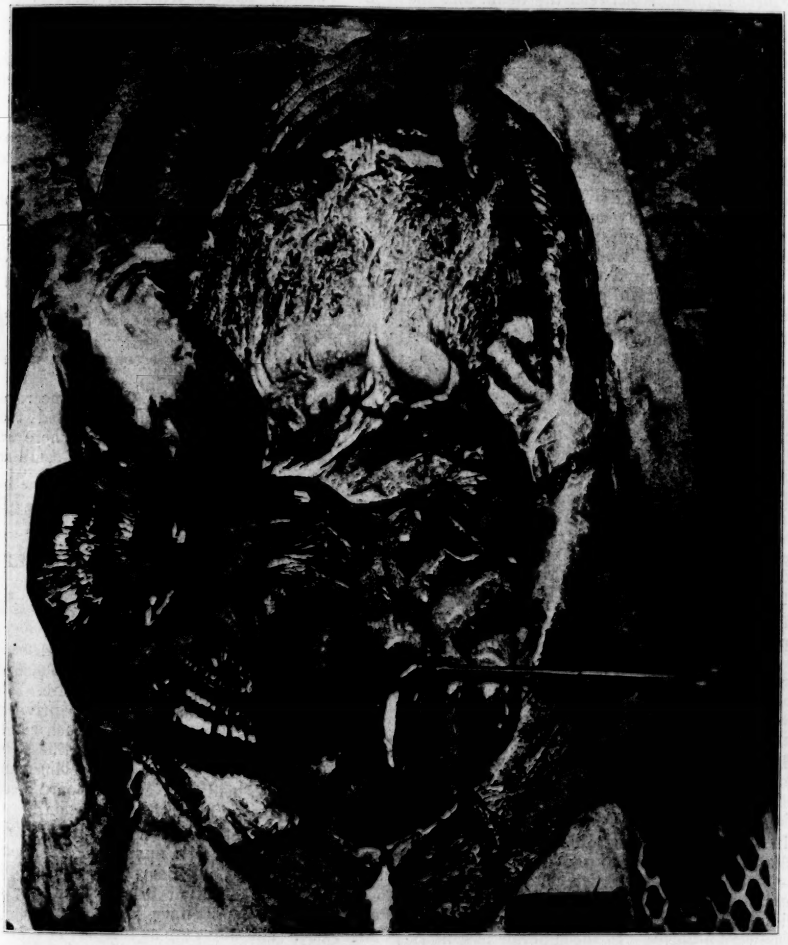
DR. RICHARDSON: That is what that would describe.

DR. YOUNG: I don't think I have anything more to say, except that whenever we see a patient with an acute abdominal emergency the

question of the rigidity of the abdominal muscles is sometimes the one thing that will differentiate between a perforation and an acute intestinal obstruction. Of course if this was a volvulus the strangulation may have come on very quickly and the symptoms be just as described here. On the other hand she had diarrhea continuing right through, which would seem to me to rule out an early volvulus. So that with that description I should have to put perforation ahead of obstruction at any time.

A PHYSICIAN: Did you say that with any strangulated hernia you find the wall not rigid?

DR. YOUNG: With intestinal obstruction from mechanical causes the abdomen is not rigid; with a strangulated hernia this is true, but there are the local signs over the hernia.



Photograph by Mr. Lewis S. Brown.
DR. OSCAR RICHARDSON.

CLINICAL DIAGNOSIS

Acute intestinal obstruction.
Mesenteric thrombosis.

ANATOMICAL DIAGNOSIS

Fibrous endocarditis of the mitral, aortic and tricuspid valves, stenosis.

Obturating thrombus of the superior mesenteric vein.
Hemorrhagic infarction of a portion of the jejunum.
Hypertrophy and dilatation of the heart.
Fibrous adhesions between the ileum and the uterus.
Chronic perihepatitis and perisplenitis.
Chronic hyperplasia of the spleen.
Chronic pleuritis.
Operation wound.

DR. RICHARDSON: A thin brownish-red fluid exuded from the operation wound. The peritoneal cavity contained a moderate amount of thin brownish-red fluid. We have to account for that of course. It came from the region of a strip of infarcted intestine. There were adhesions between the liver and diaphragm and between the spleen and the diaphragm. These adhesions extended in places to the stomach. The appendix was negative.

The first portion of the jejunum for a distance of about 70 cm. was dark purplish red, the mesentery thickened, purplish. The upper and lower margins of the infarcted strip were rather sharply marked off, the lower line of demarcation being less sharp than the upper. The wall of the jejunum beyond the portion mentioned was slightly reddened, but this soon faded out.

The large intestine was negative. At a point thirty cm. above the ileocecal valve there was a band of adhesions extending from the wall of the ileum to the posterior wall of the uterus. All told there was considerable chronic peritonitis.

There was some chronic pleuritis.

The heart weighed 440 grams,—moderate hypertrophy. The mitral, aortic, and tricuspid valves showed a moderate amount of chronic fibrous endocarditis. The mitral circumference was 7 cm., the aortic 6, the tricuspid 10.5. The coronaries were free and negative. The aorta was negative.

The spleen was quite large, 1030 grams, but all it showed was some increase in the interstitial connective tissue. There was chronic perihepatitis and perisplenitis.

In the region of the jejunum infarction the walls yielded a purplish red fluid, the mucosa was swollen, dark purplish, and the intestine contained considerable thin purplish bloody fluid. The splenic and the inferior mesenteric veins were negative. In the superior mesenteric vein there was an obturating thrombus.

DR. YOUNG: Have you any idea where this thrombus came from?

DR. RICHARDSON: That is the reason why Dr. Scudder's note is recorded. There was no definite source in the body, but if there was a twist there that is a perfectly good source.

DR. CABOT: Could the cardiac lesion by slowing the circulation be regarded as a cause of thrombosis in a vein as far off as this?

DR. RICHARDSON: There does not seem to be quite enough stasis.

DR. CABOT: There was no passive congestion anywhere else? The heart was doing its job?

DR. RICHARDSON: Fairly well apparently.

DR. YOUNG: Isn't it true that there is generally some cardiac lesion in most of these cases of mesenteric thrombus?

DR. RICHARDSON: This thrombus was in the superior mesenteric vein. I cannot say definitely. I do not remember about them in regard to that. It happened that Mr. Brown took some very successful pictures of this case which are worth looking at and are much better than anything one can say.

A PHYSICIAN: Is there anything to indicate the cause of the chronic peritonitis?

DR. RICHARDSON: Nothing definite.

A PHYSICIAN: The tubes and uterus were normal?

DR. RICHARDSON: Yes.

DR. CABOT: How do you account for the blood in the stomach?

DR. YOUNG: It was acute congestion, wasn't it?

DR. RICHARDSON: At the time of necropsy the stomach contained much undigested food material but was otherwise negative. There was bloody fluid in the first portion of the small intestine.

MISCELLANY

Three More States Accept National Board Certificate

THE State Boards of Medical Examiners of Michigan, Oklahoma and Wyoming have recently passed resolutions authorizing the acceptance of the certificate of the National Board of Medical Examiners for medical licensure. The State Board of Michigan reserves the privilege of reviewing the full credentials of each candidate for licensure who holds the certificate of the National Board. Applicants for licensure in Wyoming who hold the National Board's certificate will be given an oral examination.

This favorable action by the three state boards mentioned brings the total number of states now accepting the National Board's credential up to thirty-one, in addition to the Territory of Porto Rico and the Military Reservation of the Canal Zone. The states are as follows: Alabama, Arizona, Colorado, Delaware, Georgia, Idaho, Illinois, Iowa, Kentucky, Maine, Massachusetts, Maryland, Michigan, Minnesota, Mississippi, Nebraska, New Hampshire, New Jersey, New York, North Carolina, North Dakota, Oklahoma, Pennsylvania, Rhode Island, South Carolina, Tennessee, Texas, Vermont, Virginia, Washington, and Wyoming. In ten other states favorable legislation is now pending.—*National Board Bulletin*, Vol. II, No. 3.

THE BOSTON

Medical and Surgical Journal

Established in 1828

Published by The Massachusetts Medical Society under the jurisdiction of the following-named committee:

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SUBSCRIPTION TERMS: *$6.00 per year in advance, postage paid for the United States, $7.56 per year for all foreign countries belonging to the Postal Union.*

Material for early publication should be received not later than noon on Saturday. Orders for reprints must be sent to the Journal office, 126 Massachusetts Ave.

The Journal does not hold itself responsible for statements made by any contributor.

Communications should be addressed to The Boston Medical and Surgical Journal, 126 Massachusetts Ave., Boston, Mass.

SOME ECONOMIC FEATURES OF PRACTICE

VOLUMES have been written and many addresses have been delivered with the economic problems of present and future medical practice as the central thought.

Our rural communities are used as illustrations of failure to support doctors. The encroachment of nurses in certain fields has been regarded as a menace, the socialization of medicine through lodge practice and the invasion of the field of the general practitioner by public and voluntary health agencies have all been given prominence as explanations of unsatisfactory remuneration for the general practitioner and fears are current of worse conditions in the future. There has been a somewhat general feeling that specialists were in a class by themselves and in no danger.

There has been, at times, a rather feeble protest against one form of nursing activity which threatens one group of specialists and to some degree the ordinary doctor. We refer to the growing custom of employing nurses as anaesthetists. Men in private practice, surgical clinics and hospitals are placing the administration of anaesthetics in the hands of nurses quite generally. The argument is submitted that nurses, if properly trained, are more satisfactory as anaesthetists than most doctors because they have comparatively little interest in the details of an operation and confine themselves more exclusively to the administration of the anaesthetic and the behavior of the patient. According to common custom the administration of the anaesthetic is not under the observation of the surgeon in charge until the patient is presented to him ready for his work. The very pertinent question has been raised as to the status of the nurse when functioning as an anaesthetist; in other words, is she practicing medicine without having secured registration? If so, she is subject to prosecution and, if convicted, may be fined or imprisoned.

The argument of the employer is that the nurse under these conditions is acting as his agent and administering the drug under his direction, using as an illustration in explanation the use of morphine under the doctor's direction.

Nurses usually give ether, sometimes gas oxygen, but do not as a rule employ other anaesthetic agents. Ether is often spoken of as fool proof and the details of administration can be learned by a person of ordinary intelligence, but the apparent safety seems to be more in the immediate rather than more remote reactions. Pneumonia and disturbances of other organs are not rare complications attending etherization which may have had some relation to the methods of administration. Confidence in the safety of ether and the lower cost of the nurse service have, in all probability, been factors in the adoption of this custom.

So long as the doctor is present and controls the administration of the anaesthetic, there can be no question of the right of the nurse and doctor to follow this custom, but there is reason to doubt the right of either, if the doctor is not present. So far as the use of morphine is used as an illustration, there may be a definite deficit in the argument, for in giving orders for the use of morphine, the dose is defined by the prescriber and its repetition qualified, or at least should be. Obviously this cannot be the case with anaesthetics for the doses and the reactions vary to a marked degree in different patients and require more or less expert judgment to secure insensibility without danger.

The doubts regarding this custom are not new but the question has not been put to a legal test so that ground exists for arguments for and against this custom. Since there are honest differences of opinion, efforts should be made to ascertain the wishes of the majority of the profession, and if it is found that there is a majority opinion in favor of having nurses do this work, then both she and the doctor should be protected. If it should be decided that the nurse should not give ether because this practice should be entrusted only to physicians, the surgeons in this State who hire nurses for this purpose except in an emergency may find themselves in an unpleasant predicament because

there is a definite provision of law which provides that a registered physician may not associate himself with an unregistered person for the purpose of carrying on the practice of medicine.

To some the questions involved may seem to be almost in the category of hair splitting contentions but in the event of an unfortunate result an important issue may be made. Discussion of this subject recently seems to indicate that there is considerable interest in the minds of some doctors pro and con.

The attitude of the Examining Board of California is of interest as shown in a copy of a communication to *California and Western Medicine.*

STATE BOARD MEDICAL EXAMINERS

Sacramento, Calif., November 6, 1924.

Re: Anesthesia.

Dear Dr. Musgrave—Our legal department has held that the giving of an anesthetic by a nurse constitutes a violation of the Medical Practice Act.

In the standardization of hospitals, does your committee make any point of this important feature; i. e., is it required that anesthetics in an approved hospital must be given by one licensed under the Medical Practice Act in the State of California?

Very truly yours,

C. B. PINKHAM, M.D., *Secretary-Treasurer.*

The JOURNAL will welcome an expression of opinion.

LEGISLATIVE NOTES

The Hearing on House Bills Nos. 665 and 531

THE first is the bill drafted by Dr. S. B. Woodward and the other by the opponents of compulsory vaccination.

The hearing on Dr. Woodward's bill was conducted by its author who presented a clear and logical resume of the smallpox and vaccination situation. His compilation of facts is complete and his arguments were free from flaws, cogent and appealing.

Dr. Eugene R. Kelley, Commissioner of Health, registered the approval of the State Department of Public Health of Bill No. 665 and opposition to Bill 531. He then introduced Dr. C. C. Pierce of the U. S. Public Health Service, who has had an extensive experience in dealing with state officials and others during smallpox epidemics. His experience was convincing testimony to the soundness of Dr. Woodward's arguments. One especial feature of his address was a report of the actual expense of meeting smallpox epidemics as contrasted with the cost of vaccination.

We have been promised an abstract of his speech.

Dr. Bigelow, representing the Massachusetts Medical and the Massachusetts Homoeopathic Societies, registered approval of Dr. Woodward's bill and opposition to the other. The Friends of Medical Progress, the Boston Health Department and the Boston School Department also appeared in favor of Dr. Woodward's bill.

Mr. Nunn, Secretary and Executive Officer of the Medical Liberty League Incorporated, conducted the hearing for the opposition to Dr. Woodward's bill and in favor of Bill No. 531.

He felt that the question is not whether vaccination prevents smallpox but rather the logic of compelling school children to be vaccinated contrary to the wishes of parents. He introduced two witnesses who felt that vaccination was responsible for the death of a member of the family of each of these speakers. The certificates of death, however, were recorded as infantile paralysis and meningitis. The comparatively near association of these deaths with a previous vaccination led to the supposition that vaccination was responsible for the deaths.

Dr. Padelford of Fall River presented the same arguments which he has used before, most of which are based on comparatively ancient history.

He persisted in using the Philippines as an illustration, although Dr. Leonard Wood has given an adequate explanation of the epidemic of 1917 and 1918 and shown that since then vaccination has practically freed the Islands of smallpox.

Dr. Benjamin White was called on to refute the charges that vaccine matter is carelessly and improperly prepared.

If the legislature can be diverted from the adoption of measures which will bring more general protection from smallpox after this hearing there is little hope of an intelligent treatment of this subject. Not one of the arguments of the proponents for Dr. Woodward's bill was refuted or even weakened by the contentions of the opponents.

The attitude of the opponents warrants the feeling of pity for their inability to accept the scientific demonstration of the efficacy of vaccination. We must conclude that they are unable to approach the subject in a disinterested and judicial state of mind.

MISCELLANY

Stringent Health Rule Enforced by Utah City

THAT an innovation in public health measures is now being enforced in Salt Lake City is disclosed by an article that will appear in the January issue of *Venereal Disease Information,* a magazine published by the Division of Venereal Diseases of the United States Public Health Service.

According to this magazine, the Salt Lake City authorities have been making a systematic effort to quarantine all men and women found under circumstances which give rise to a reasonable suspicion that they have a venereal disease. Under this rule, any situation which will warrant a responsible inference that the persons are engaged in prostitution or promiscuity provides legal sanction for compelling an examina-

tion, and, if an infection is shown, to establish a quarantine and enforce treatment. Both men and women are subject to the working of the measure.

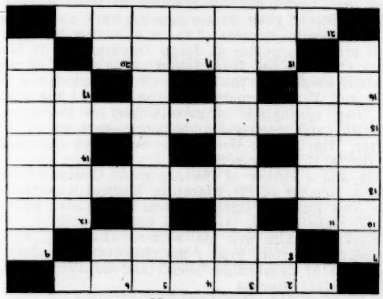
This rule was recently put into effect by the board of health of Salt Lake City acting in coöperation with the Utah state board of health. —*United States Public Health Service.*

Child Cripples

THERE are about 264,000 crippled children in the United States, according to an estimate in the recently published "The Care, Cure, and Education of the Crippled Child," by the International Society for Crippled Children.

Infantile paralysis ranks first in the list of causes of crippling conditions in children, "surgical" tuberculosis second, congenital deformities third, and rachitic deformities fourth, it is said.

A STORMY CONVALESCENCE



HORIZONTAL

1. The Greek word for "plug."
8. An aphrodisiac.
10. A symbol for myopic astigmatism.
12. A suffix signifying "capable of."
13. Tendency to putrefaction.
14. A suffix signifying "pertaining to."
15. An agent that destroys mites.
16. The Egyptian sun god.
17. A suffix forming the comparative degree.
18. Soil.
21. Latin for "bath."

VERTICAL

2. The author's product.
3. Pertaining to schizomycetes.
4. A chemical suffix.
5. The bane of bacteria.
6. A famous university (abbr.).
7. The useful laxative.
9. A combining form for glycerin.
11. An Oriental balsam.
12. To happen (obs.).
18. A Babylonian deity, the healer of the sick.
19. Registered nurse (abbr.).
20. A man's name (abbr.).

(*Solution will appear in next week's issue.*)

Appointments at Harvard Medical School

THE following appointments were made at the Harvard Medical School on February 9th:

Reappointment for six months from Feb. 1, 1925: John Verner Leech, A.M. Assistant in Comparative Anatomy. Albert Aurelius Horner, M.D. Assistant in Tropical Medicine.

New appointment from June 1, 1925, to Sept. 1, 1925, and from Sept. 1, 1925, to Sept. 1, 1926: Jack Henry Sandground, M. Sc. Instructor in Tropical Helminthology. Department of Tropical Medicine.

New appointment for six months from February 1, 1925: Joseph Hamilton Burnett, M.D. Assistant in Anatomy. Thomas Raymond Haig, M.D. Assistant in Anatomy.

New appointment for one year from September 1, 1924: Urosh S. Rusitchitch, M.D. Research Fellow in Pediatrics.

Errata

IN the account of the meeting of the Boston Medical History Club two errors occur. The spelling of the names of Doctors Welch and Futcher was incorrect.

Dr. Downing to Visit Skin Clinics

DR. JOHN GODWIN DOWNING, 784 Beacon Street, Boston, and medical inspector of the Boston Health Department, sailed February 15 on the Pilgrimage to Rome, headed by His Eminence, Cardinal O'Connell. After visiting Rome, Dr. Downing will visit the celebrated skin clinics in Vienna, Paris, Berlin and London.

Dr. Downing is a member of the Michael Perkins Post, American Legion, and was an officer of the United States Navy, in 1917 to 1921. He is a graduate of Boston College, (A.B.) 1911, and Harvard Medical School, (M.D.) 1915.

Results of the National Board Examinations

OF the 60 candidates who took the complete examination in Part II in September four of the ten highest candidates are graduates of the Harvard Medical School and one of the Boston University School of Medicine.

In the September examination, Part I, five of the ten highest candidates are students of the Harvard Medical School.

The leader in Part II was Louise D. Schweinitz, a student at Johns Hopkins University, Medical Department.

The leader in Part I was George K. Speare who is a student of Harvard Medical School.

"Heart Disease Has Supplanted Tuberculosis as the Greatest Man-Killer"

THIS assertion was made by Dr. Robert H. Halsey, of New York, in a paper presented to the American Association for the Advancement of Science at its last annual meeting in Washington, D. C. The assertion is further substan-

tiated by Dr. Stewart R. Roberts in a paper recently read before the Southern Medical Association in New Orleans, and by figures just given out for the year 1923 by the United States Department of Commerce. Dr. Roberts not only places heart disease as the most deadly destroyer of human life, but he also points to syphilis as one of the greatest causes of heart disease. It was said at the meeting that syphilis is responsible for 52 per cent. of all heart troubles.

According to statistics compiled by the Department of Commerce for the registration area, comprising 87.6 per cent. of the population of the United States, heart disease claimed 170,033 lives in 1923, as compared to 105,680 deaths caused by pneumonia and 90,732 by tuberculosis. Syphilis is credited with a toll of 16,811 deaths.

"If the above figures are correct," says the Division of Venereal Diseases of the United States Public Health Service, "syphilis may also be held responsible for 52 per cent. of all the deaths resulting from heart trouble, or 88,417 fatalities. When this sum is added to the 15,811 deaths credited to syphilis by the census figures, the result is a total of 104,228 deaths caused by syphilis directly or indirectly, and this total is arrived at without taking into consideration deaths caused by some other diseases which are in many cases a result of syphilitic infection. Experiments recently made in Paris would seem to indicate that 76 per cent. of syphilities have heart trouble. Out of autopsies on 155 known syphilities there were 115 cases in which vascular lesions of the heart were found.—*United States Public Health Service.*

RECENT DEATHS

HEATHFIELD.—Dr. Frank Edward Heathfield—Harvard Medical School, 1924—died after a brief illness at the Germantown Hospital, Germantown, Penn., on February 4, 1925.

Dr. Heathfield was the son of Albert E. Heathfield of Smeath, near Ashford, in Kent, England. He graduated with the degree of A.B. in 1920 from Wheaton College, Wheaton, Ill., and from the Harvard Medical School in 1924.

Dr. Heathfield's death was particularly sad because he entered on an interneship at the Germantown Hospital on January 1, 1925. Only 15 days later he developed a sore throat, from which was cultured a hemolytic streptococcus. After several days in bed he went on duty again, only to be forced to bed with chills and fever. A blood culture showed the same organism—hemolytic streptococcus. He knew the seriousness of his disease and made a brave fight. On February 4 he died. The funeral was held February 6 at St. Mary's Church, Ardmore, Penn. He was buried in Arlington Cemetery, near Landsdowne, Penn. His classmate, Mr. Fisher, and Dr. C. T. Hunter and Dr. H. E. Appel were among the pallbearers. His Harvard classmates sent a wreath with the following inscription:

Frank E. Heathfield
with the
Love and Affection of His Classmates
to whom
From "falling hands" the torch is thrown.
May we "hold it high!"

HOLLAND.—Dr. William Timothy Holland died at his home in West Roxbury, February 10, 1925, aged 47. He had been ill since September, and pneumonia developed two days before his death.

He was born in Winchester and was graduated from Tufts College Medical School in 1898. He was a Fellow of the Massachusetts Medical Society from 1898 to 1910, when he moved from Brookline to West Roxbury. He was chairman of the public health committee of the West Roxbury Citizens' Association, a member of the West Roxbury Post, A. L., the Military Order of the World War, John J. Williams Council, K. C., and was a supporter of St. Vincent de Paul's Society and other charitable organizations. Surviving are his widow, Mrs. Lillian Holland; a daughter, Miss Helen G. Holland, who is a teacher at the Girls' Latin School, and a son, Paul Holland, a student at Harvard.

STEVENS.—Dr. Henry Burt Stevens, a Fellow of the Massachusetts Medical Society, died at his home in West Roxbury, January 31, 1925, of pneumonia.

He was born in Norwich, Conn., January 6, 1870. After graduating from the Harvard Medical School in 1894 he served as interne in the Boston City Hospital and the Backus Hospital in his native city, and then began general practice in West Roxbury. About 1909 he gave up his general work and limited his practice to diseases of the eye, having taken several graduate courses at Johns Hopkins and in New York City. In the years following he served as ophthalmic surgeon at the Boston City Hospital and the Eye and Ear Infirmary. At the time of his death he was ophthalmic surgeon-in-chief at the former hospital and assistant ophthalmic surgeon at the latter. During the World War he served as captain in the M. C. at Camp Devens.

He was a Fellow of the American College of Surgeons, member of the American Medical Association, the New England Ophthalmological Society, various social clubs and the American Legion.

Surviving him are his widow, who was Bertha Reese of Scranton, Penn.; his daughter, Mrs. Joseph Erickson of Cambridge, Mass., and an adopted son, Joseph Kent Stevens.

OBITUARY

IN MEMORIAM

Andrew James Halpin

It is with deep sorrow that the fellows of the Middlesex North District Medical Society record the death of their beloved associate.

Dr. Halpin was born in Lowell in 1864 and lived in that city during his entire life, being engaged actively in the practice of medicine from 1890 until his death.

He was a graduate of the Lowell High School, a graduate of McGill University with the degree of Bachelor of Arts and a graduate of Harvard Medical School in 1889.

He was a man of decided characteristics. He was diligent and studious to a fault. This quality was so markedly developed that he never spared himself in the least when attending to his professional duties, and he devoted himself all too faithfully and unsparingly to his life work.

Being endowed by nature with a mind of distinguished excellence, his whole-hearted zeal could not fail to bring him to the foremost rank in his profession.

Dr. Halpin was modest and retiring, and he cared not for general society or for public life, but he was loved, as are very few men, by his friends who were many, and he will be greatly missed and deeply mourned by all.

And deeply will be mourned by the Middlesex North District Medical Society.

To the bereaved family we extend our most profound sympathy.

LEONARD HUNTRESS,
EDWARD J. WELCH,
WILLIAM M. COLLINS,
Committee.

CORRESPONDENCE

AN ACCOUNT OF INSULIN REACTION

226 Marlboro Street, Boston,
January 30, 1925.

Editor, Boston Medical and Surgical Journal:

The following is an interesting account of an Insulin reaction in Case No. 104:

The patient is a young lady of 20, a senior in college, a mildly severe diabetic since 1918 and one of our first patients to have Insulin after its discovery. Her usual dose of Insulin is 60 units daily—22 units before breakfast, 16 units before luncheon and 22 units before dinner. Only occasionally has she noticed the least signal of an Insulin reaction, and always in the past she has taken the juice of an orange. Her diet is perfectly controlled and only rarely shows a trace of sugar in the urine, and her blood sugar remains between 0.10 per cent. and 0.14 per cent.

During the holidays a trace of sugar appeared, and without supervision, but according to custom, she reduced her carbohydrate from 10 grams to 20 grams in 24 houra. On the evening of the 6th the 22 units of Insulin was taken as usual and, shortly after this, dinner was served. During the meal members of the family noticed that there was some exhilaration and a tendency to repeat during a story she was telling. Immediately after dinner a young lady friend was told that she appeared unnatural and another member of the family was told that things seemed to be blurred.

At about 8 o'clock, some few moments after finishing the meal, her actions became definitely strange and aid, when offered, was resented. However, she was finally persuaded to lie down. It was feared that an Insulin reaction might be taking place, so, approximately at 8.30, one of her physicians was called. He arrived shortly after 9 o'clock and found the patient unable to coöperate and entirely flaccid, with slow respiration and a pulse of 60, which, however, was fairly strong. At this time there was absolute inability to coöperate on the part of the patient. A few teaspoonsful of glucose were forced between her teeth and her blood sugar was taken, which proved later to be 0.033 per cent.

Between 9.30 and 10 she was somewhat aroused, but very resentful when the glucose was forced upon her. At 10 o'clock a cup of coffee and 10 grams of carbohydrate was given her and she was permitted to rest.

At 11 o'clock she looked and acted perfectly normal in every way, but had no memory of events from the time that she had risen from the dinner table.

The interesting feature about this reaction is that it took place in a young woman who is a perfectly trained diabetic, and that the Insulin reaction crept upon her so gradually that there was no realization of its approach. On every previous occasion there had been a warning. This reaction gave none of the cardinal warning symptoms.

Another patient of mine, a very intelligent doctor, who has had a long-standing diabetes, on two occasions has described similar reactions, and on one occasion a temporary psychosis developed and continued for two hours.

Sincerely yours,
B. H. RAGLE.

RUPTURED ECTOPIC PREGNANCY

261 Central Park West, New York City,
February 10, 1925.

Editor, Boston Medical and Surgical Journal:

In the BOSTON MEDICAL AND SURGICAL JOURNAL, December 11, 1924, there appears a paper by Anthony Corvese on "Shoulder Pain as an Indication of Ruptured Ectopic Pregnancy," in which piquant reference is made to a publication of mine that appeared in the *J. A. M. A.*, April 14, 1923 (Vol. 80, pp. 1050-1052). Corvese states that "a few days before Rubin published his article I had operated on a woman with a ruptured ectopic pregnancy in which the shoulder and neck pain was so marked before the operation that it gave rise to considerable discussion as to its cause, etc. The sudden disappearance of the pain immediately after the operation stimulated me to an investigation." Corvese is evidently much disappointed that his case should come to his notice but a few days before my publication and he goes on to show that, although "none of the books on gynecology mention this symptom," so many others had preceded me that "it is *simply* absurd for anyone at the present day to put it forth as a new and original idea."

While I was engaged in studying the significance of shoulder pains in uterine insufflation my attention was called to the occurrence of shoulder pains in patients with ruptured ectopic pregnancy. *The first observation was made on a ward patient in October, 1921.* This case was presented by me at the regular clinical conference of the gynecological staff of the Mount Sinai Hospital in November, 1921.

It is certainly gratifying that the observation reported by me with the title "Sudden acute pain in the shoulders associated with acute pelvic pain in woman, a symptom of ruptured ectopic pregnancy, indicating subphrenic blood extravasation (subphrenic hemoperitoneum)," is one that has been borne out by the experience of others, which, of course, would tend to establish it as a differential point in diagnosis; and I am under obligations to Corvese for bringing together the important contributions dealing with this specific point in diagnosis.

He goes on to say, however: "Herzfeld makes careful reference to those who had preceded him in the investigation of the relation between phrenic shoulder pains and ruptured ectopic gestation, but in this respect he is not imitated by Rubin, who, a few weeks later, made public in this country what he leaves us to assume are entirely independent and original observations upon the subject." Herzfeld's paper appeared, according to Corvese, in the *Zentralblatt f. Gynäk.*, March 31, 1923, 47, No. 13, 517. My paper appeared in the *J. A. M. A.* April 14, 1923—just two weeks later—and was submitted for publication at least two months before.

Moreover, my paper was submitted to D. Appleton & Co. with the manuscript of my monograph, "Symptoms in Gynecology and Their Interpretation," in November, 1922, and it was written long before then.

although published November, 1923. Hans Dawes' publication appeared September, 1922, and I had not seen it. It would appear from Corvese's article, however, that Behan in 1914 made the first observation that the pain in extra-uterine pregnancy in some cases is referred to the shoulders and gave its true explanation. I do not know whether some one long before him may not have called attention to this clinical symptom-complex.

No claim to priority was intended by my publication. After my paper appeared a number of colleagues told me they had been observing this phenomenon. It may not be unfair to suppose that through the impetus of transuterine insufflation and its attending shoulder pains many others in very recent years may have struck upon the relationship between shoulder pains and ruptured ectopic pregnancy. That it had not been known generally must be evident from the absolute lack of reference in the textbooks dealing with this subject. The clinical observation is strengthened by the fact that it had been reported by others before my own publication, and since then it has become established on firm ground.

Very truly yours,
I. C. RUBIN.

NEWS ITEM

NEW DIRECTOR OF THE DIVISION OF CHILD HYGIENE, CHILDREN'S BUREAU

Dr. Martha M. Eliot, a graduate of Johns Hopkins University Medical School and a member of the faculty of the Department of Pediatries, Yale University School of Medicine, has been appointed director of the division of child hygiene of the Children's Bureau. During the past year Dr. Eliot has been in charge of a demonstration of methods for the control of rickets in young children, which the Pediatrics Department of Yale University School of Medicine has been conducting in coöperation with the Children's Bureau, in New Haven.

REPORTS AND NOTICES OF MEETINGS

A PHYSIOLOGICAL CONFERENCE will be held Wednesday, March 4, in the Bowditch Library, Building C of the Harvard Medical School, at 4 P. M. Mr. V. A. Pertzoff will speak on "The Effect of Rennin Upon Caffeine."

Essex South District Medical Society

THE Regular Meeting will be held at the Essex Sanatorium, Middleton, Wednesday, March 4, 1925, at 5 P. M. There will be a clinic by members of the Staff. The following cases will be shown and discussed:

1. Differential Diagnosis between Pulmonary Tuberculosis and Carcinoma.
2. Progressive Infectious Arthritis.
3. Tuberculosis complicated by Diabetes.
4. Primary Round Cell Carcinoma of the Lung.
5. Artificial Pneumonothorax Treatment.
6. Differential Diagnosis between Tuberculosis and Hyperthyroidism.

At 6 P. M., Moving Pictures:

1. Diphtheria.
2. Smallpox.

At 7 P. M. Dinner, followed by the speaker of the evening, Dr. Frederick T. Lord of Boston, who will speak on "Certain Aspects of Diseases of the Bronchi, Lungs and Pleura."

R. E. STONE, M.D., *Secretary*,
Beverly, Mass.

Massachusetts State Nurses' Association

NORFOLK AND SUFFOLK COUNTY BRANCHES

THE monthly meeting will be held at 636 Beacon Street, Boston, Thursday, February 26, 8:15 P. M.

Lecture: "What Nurses Should Know About Tuberculosis."

Lecturer: Dr. John B. Hawes, 2nd, Boston. All nurses invited.

MARY ALICE MCMAHON, R. N., *Secretary*.

The Annual Congress on Medical Education, Medical Licensure, Public Health and Hospitals, March 9, 10, 11 and 12, 1925, Congress Hotel, Chicago

PRELIMINARY PROGRAM

COUNCIL ON MEDICAL EDUCATION AND HOSPITALS

MONDAY, MARCH 9, 1925

MORNING SESSION, 9:30 A. M.

1. Opening Remarks—Ray Lyman Wilbur, M.D., President of Stanford University.
2. The State University and Medical School——Walter A. Jessup, Ph.D., LL.D., President of the State University of Iowa, Iowa City.
3. The Founding of a Medical College in a New Province—Henry M. Tory, D.Sc., LL.D., President of the University of Alberta, Edmonton.
4. Symposium—Twenty-five Years' Progress in Medical Education—(a) Entrance Requirements, Grading of Students, Promotion, etc. —John M. Dodson, M.D., Former Dean of Rush Medical College, Chicago. (b) Buildings, Physical Equipment, Finances and Faculty—Burton D. Myers, M.D., Assistant Dean of Indiana University School of Medicine, Bloomington. (c) Anatomy—Clarence M. Jackson, M.D., University of Minnesota Medical School, Minneapolis, (d) Biochemistry —Albert P. Mathews, Ph.D., University of Cincinnati College of Medicine.

AFTERNOON SESSION 2:00 P. M.

4. Symposium—Twenty-five Years' Progress in Medical Education (Concluded). (e) Physiology and Pharmacology—Charles C. Guthrie, M.D., University of Pittsburgh School of Medicine. (f) Pathology and Bacteriology —James Ewing, M.D., Cornell University Medical College, New York. (g) Medicine and the Medical Specialties—Samuel W. Lambert, M.D., Emeritus Dean of Columbia

University College of Physicians and Surgeons, New York. (h) Surgery and Surgical Specialties—William D. Haggard, M.D., Vanderbilt University School of Medicine, Nashville. (i) Obstetrics and Gynecology—Reuben Peterson, M.D., University of Michigan Medical School, Ann Arbor.

TUESDAY, MARCH 10, 1925

MORNING SESSION, 9:30 A. M.

1. Results of Improved Medical Education in the Government Medical Services. (a) The Army—Merritte W. Ireland, M.D., Surgeon-General of the United States Army. (b) The Navy—Edward R. Stitt, M.D., Surgeon-General of the United States Navy. (c) The Public Health Service—Hugh S. Cumming, M.D., Surgeon-General of the United States Public Health Service.
2. Coöperation in Medical Education—Charles R. Bardeen, M.D., Dean of the University of Wisconsin Medical School, Madison.
3. Correlation of Teaching Between the Laboratory and Clinical Departments of the Medical School—O. H. Perry Pepper, M.D., Assistant Professor of Medicine of University of Pennsylvania Medical School, Philadelphia.
4. Conditions of Medical Practice in Canada—A. Primrose, M.D., Dean of the University of Toronto Faculty of Medicine.

AFTERNOON SESSION, 2:00 P. M.

Medical Service in Rural Communities

1. Educational Conditions in Rural Communities—George F. Zook, Ph.D., Specialist in Higher Education of the United States Bureau of Education.
2. Present Conditions and Tendencies in Rural Affairs of America—William L. Bailey, Ph.D., Professor of Sociology, Northwestern University, Evanston, Illinois.
3. Distribution of Physicians in the United States—William Allen Pusey, M.D., President of the American Medical Association, Chicago.
4. On the Report of the General Education Board—Raymond Pearl, Ph.D., Professor of Biometry and Vital Statistics, Johns Hopkins University School of Hygiene and Public Health, Baltimore.
5. The Situation in Minnesota—Elias P. Lyon, M.D., Dean of the University of Minnesota Medical School, Minneapolis.

FEDERATION OF STATE MEDICAL BOARDS

WEDNESDAY, MARCH 11, 1925

MORNING SESSION, 9:30 A. M.

1. Symposium—Essentials of an Adequate Examination. (a) Principles and Methods Underlying Adequate Achievement Measures—Donald G. Paterson, A. M., Professor of Psychology of the University of Minnesota, Minneapolis. (b) Fundamental Medical Sciences—Frederick C. Waite, Ph.D., Professor of Histology and Embryology of Western Reserve University School of Medicine, Cleveland. (c) Clinical Medical Sciences—David L. Edsall, M.D., Dean of Harvard Medical School, Boston. (d) Medical Licensure—Irvin D. Metzger, M.D., President of the Pennsylvania Board of Medical Education and Licensure, Pittsburgh. (e) Written versus Practical—Walter L. Bierring, M.D., Secretary of the Federation of State Medical Boards, Des Moines. Discussion opened by Elias P. Lyon, M.D., Dean of the University of Minnesota Medical School, Minneapolis. Horace D. Arnold, M.D., Boston. Samuel W. Welch, M.D., Chairman of the Alabama State Board of Medical Examiners, Montgomery.
1. Symposium—Essential Principles of a Medical Practice Act. (a) Functions of a Medical Practice Act—William C. Woodward, M.D., LL.M., Secretary of the Bureau of Legal Medicine and Legislation, American Medical Association, Chicago. (b) Eligibility for License. (a) Essential Qualifications. (b) Methods of Determining—Charles B. Pinkham, M.D., Secretary of the California State Board of Medical Examiners, San Francisco. (c) Methods of Enforcement. (a) (Criminal Prosecution. (b) Quo Warrento. (c) Injunction Procedure. (d) Revocation of License—H. M. Platter, M.D., Secretary of the Ohio State Medical Board, Columbus. (d) Diagnosis and Treatment—Harry Eugene Kelly, Esq., of the Chicago Bar. Discussion: Augustus S. Downing, Ph.D., Assistant Commissioner and Director of Professional Education of the University of the State of New York, Albany. Thomas J. Crowe, M.D., Secretary of the Texas Board of Medical Examiners, Dallas. Elbridge M. Shanklin, M.D., President of the Indiana State Medical Association, Member Indiana Board, Hammond.

AMERICAN CONFERENCE ON HOSPITAL SERVICE

THURSDAY, MARCH 12, 1925

MORNING SESSION, 9:30 A. M.

1. Annual Report of the Hospital Library and Service Bureau—Miss Donelda R. Hamlin, Director of the Hospital Library and Service Bureau.
2. The Extension of Hospital Privileges to All Practitioners of Medicine—S. S. Goldwater, M.D., President of the American Conference on Hospital Service.
3. Hospital Facilities and the Medical Profession in the United States (Statistical Summary)—Mr. Homer F. Sanger of the Council on Medical Education and Hospitals of the American Medical Association.

4. Educational Opportunities of the Open Hospital—Malcolm T. MacEachern, M.D., Associate Director of the American College of Surgeons.
5. Educational Opportunities of the Closed Hospital—Arthur C. Bachmeyer, M.D., President-Elect of the American Hospital Association.

BUREAU OF HEALTH AND PUBLIC INSTRUCTION

AFTERNOON SESSION, 2:00 P. M.

The Medical and Health Education of the Public

1. By Means of Bulletins and Journals—Herman N. Bundesen, M.D., Commissioner of Health, City of Chicago.
2. By Means of Newspapers—Morris Fishbein, M.D., Editor of the Journal of the American Medical Association.
3. By Means of Addresses and Radio Talks—Hugh S. Cumming, M.D., Surgeon-General of the United States Public Health Service.
4. By Means of Health Exposition and Exhibits—William A. O'Brien, M.D., of the Department of Pathology, University of Minnesota Medical School, Minneapolis.
5. By Means of Periodic Examinations of Apparently Healthy Persons—Joseph L. Miller, M.D., Clinical Professor of Medicine of Rush Medical College, Chicago.

These meetings are of great interest to all physicians.

Program of the Thirty-Fifth Annual Meeting of the Association of American Medical Colleges To Be Held at Boston, Mass., March 5, 6 and 7, 1925

THURSDAY, MARCH 5, BOSTON CITY HOSPITAL, 9:30 A. M.

Medical Sociology and Environment Medicine—Charles P. Emerson, Dean University of Indiana School of Medicine.

Bearing of Neuropsychiatry on Public Health Problem—Albert M. Barrett, Professor of Neuropsychiatry, University of Michigan Medical School.

Education in Preventive Medicine in Regular Curriculum—Haven Emerson, Professor of Public Health Administration, Columbia University College of Physicians and Surgeons.

Teaching of Preventive Medicine—Samuel R. Haythorn, Professor of Pathology and Bacteriology, University of Pittsburgh School of Medicine.

2:00 P. M.

Address of President: The Future Practitioner—Ray Lyman Wilbur, President Stanford University.

Correlation in the Curriculum—Bernard F. McGrath, Professor of Principles of Surgery, Marquette University School of Medicine.

Teaching of Obstetrics—J. M. H. Rowland, Dean University of Maryland School of Medicine.

Teaching of Physiotherapeutic Measures—W. H. MacCracken, Dean Detroit College of Medicine and Surgery.

Reading of New Amendments to By-Laws.

FRIDAY, MARCH 6, MORNING

Practical Demonstrations in Medical Teaching—Harvard Medical School, Boston University School of Medicine, Tufts College Medical School.

ROUND TABLE CONFERENCE, HARVARD MEDICAL SCHOOL, 2:00 P. M.

Remarks by A. Lawrence Lowell, President Harvard University.

The Honors Course—E. P. Lyon, Dean University of Minnesota Medical School.

"Full Time"—Frederick T. van Beuren, Jr., Associate Dean Columbia University College of Physicians and Surgeons.

The Handling of the Superior Student—David L. Edsall, Dean Harvard Medical School.

Coöperative Education in Medicine—Newton Evans, President College of Medical Evangelists.

The Curriculum—Fred C. Zapffe, Secretary Association of American Medical Colleges.

Importance of Physical Plant in the Correlation of Teaching Medicine—R. R. Higgins, Dean University of Pittsburgh School of Medicine.

Better Correlation of Teaching of Science Branches with Clinical Subjects—H. C. Tinkham, Dean University of Vermont College of Medicine.

Administration of Hospital Medical School—Thmas Ordway, Dean Albany Medical College.

Promotion of Friendliness Between Faculty and Students—Walter L. Niles, Dean Cornell University Medical College.

An Initiatory Course for Freshmen—Stephen Rushmore, Dean Tufts College Medical School.

Greater Boston Medical Society

EAR, NOSE AND THROAT NIGHT

A meeting of that society was held Monday, January 12, 1925, in the Boston Medical Library.

PROGRAM

8:45 p. m.

1. Louis M. Freedman, M. D. "Mastoid Infections; Diagnosis and Operative Treatment."

The object of this paper is to call attention to the necessary elements which help to decide

whether a given mastoid condition is operable. Absolute drainage is required and the certainty that the process is not extending to areas which are a source of danger as to life. That also the preservation of hearing is an important deciding factor, functionally and economically. That early bringing the process to the surface is most advisable and comparable to the position we hold in relation to treatment of the inflammatory appendix. That it is safer to err on the side of putting an end to the process by bringing it to the surface sooner, than to allow it to get beyond control.

Individual judgment of each case is necessary and for this each sign and symptom should be studied carefully in cooperation with the Internist and Neurologist, making full use of all tests at our command.

2. Louis Arkin, M. D. "Tonsillectomy and Adenotomy. A Study of 1000 Cases." (This paper will be published in full.)

3. Harry P. Finck, M. D. "Foreign Bodies in the Respiratory Passages and Oesophagus."

Dr. H. P. Finck spoke on, "Foreign Bodies of the Respiratory Tract and Oesophagus." Emphasis was placed on:

1. Symptomatology of acute versus chronic cases.

2. Diagnosis both by clinical examination and X-ray.

3. Treatment, including description of technique and preparations for operations.

4. Increasing value of tracheotomy in emergency cases.

Discussion was opened by Drs. Philip E. Meltzer, Louis E. Wolfson, Samuel Cline, and Joseph Prenn.

American Heart Association Annual Meeting

THE first Annual Meeting of the American Heart Association was held at the New York Academy of Medicine, Monday afternoon, February 2nd, 1925. Dr. Lewis A. Conner, the President, reviewed briefly the progress of the development of the Association and commented on the need for such an organization and the wide-spread interest which its formation had developed. Dr. Haven Emerson, Chairman of the Committee on Membership, described the methods employed to reach all parts of the United States and Canada; regional agents covering one or more states have been appointed and already 37 states are represented in the membership of the Association. The following program for the coming year was adopted:

1. *Maintenance of a Central Office.* As a headquarters through which all the work of the Association may be organized and directed. This office is already established but the personnel must be increased to keep pace with the demands which are increasing daily.

2. *Organization and Membership.* One of the first and most important steps in the development of the organization of the Association is that of securing contacts with all parts of the United States and Canada. This is to be accomplished by appointing regional representatives and obtaining a large and widely distributed membership.

3. *Educational Work.* Distribution of literature. Publication of a monthly Bulletin. Lectures. Loaning of charts and lantern slides. Participation in Public Meetings of medical and health organizations.

4. *Field Work.* One or more workers with a knowledge of the most recent and approved methods for the prevention of heart disease and the organized care of cardiac patients, who shall be available to be sent to different sections of the country to encourage the establishment of new centers, demonstrate the methods found to be effective, and aid in solving local problems as they arise.

All present members of the Advisory Council were reëlected and the following were elected as new members of that body:

Mr. Alfred Aiken, Dr. Lindsay R. Williams, Dr. John Wyckoff, Dr. Robert Babcock, Dr. Joseph Miller, Dr Henry Sewall, Dr. Emmet F. Horine, Dr. Frank N. Wilson, Dr. Ross V. Patterson, Mr. William H. Hayes, Mr. Alfred B. Meacham, Mr. Herbert L. Kelly, Mr. Clayton M. Michael, Mrs. Fritz B. Talbot, Dr. Walter Wilson, Dr. S. A. Levine, Dr. Burton Hamilton, Dr. F. Janney Smith.

Those serving up to the time of the Annual Meeting as a temporary Board of Directors were reëlected as a permanent Board of Directors.

At a meeting of the Board of Directors immediately following the Annual Meeting, the following officers were elected:

President—Dr. Lewis A. Conner.
Vice-President—Dr. James B. Herrick.
Secretary—Dr. Robert H. Halsey.
Treasurer—Dr. Paul D. White.

Acting Executive Secretary—Miss M. L. Woughter.

The Representatives of the Massachusetts General Hospital in Charge of the Program at the Stated Meeting of the New York Academy of Medicine

ON February 19 the stated meeting of the Academy was given over to representatives of the Massachusetts General Hospital. Dr. J. H. Means presented the introductory remarks, Dr. F. T. Lord spoke on "Certain Aspects of Pulmonary Suppurative Lesions," Dr. F. Fremont-Smith's subject was "Diagnostic Value of Cerebro-spinal Fluid in Central Nervous System Infections" and Dr. J. C. Aub spoke on "Lead Poisoning."

SOCIETY MEETINGS

Essex North District Medical Society

May 6, 1925. Annual meeting at Lawrence.

Franklin District Medical Society

The meetings of the Franklin District Medical Society will be held on the second Tuesday of March and May.

Hampden District Medical Society

Meeting to be held on the third Tuesday in April.

Hampshire District Medical Society

The meetings will be held the second Wednesday of March and May.

Middlesex East District Medical Society

Wednesday, March 18. Harvard Club. Dr. John H. Cunningham, "Urinary Retention: Its Significance and Treatment."
Wednesday, April 15. Harvard Club.
Wednesday, May 13. Colonial Inn, North Reading.

Middlesex North District Medical Society

April 29, 1925.

Middlesex South District Medical Society

Winter Schedule—The plans for winter meetings of the Society include the stated meeting in April, two hospital meetings, and five meetings to be held in conjunction with the Suffolk District Medical Society and the Boston Medical Library (two surgical, two medical, and one general).

Norfolk District Medical Society

March 31, 1925. Tufts College Medical School. This meeting given over to Drs. Leary and Watters for the purpose of giving us a medical examiners' talk.

Norfolk South District Medical Society

Meetings will be held the first Thursday of each month to May, inclusive, at 12 noon, at the Norfolk County Hospital, South Braintree.

Suffolk District Medical Society

March 25. Medical Section, in association with the Middlesex South District Medical Society. "The Treatment of Pneumonia," Dr. Edwin A. Locke.
April 29. Annual meeting. "Hypertension and Longevity," Dr. Harold M. Frost.

Worcester District Medical Society

March 11, 1925. St. Vincent's Hospital, Worcester. Papers will be read by the members of the hospital staff.
April 9, 1925. Subject and speaker to be announced.
May 14, 1925. Annual meeting.

If you desire further information in regard to these meetings write to the Secretaries of the District Medical Societies (listed on page ix of the Advertising Section). The Massachusetts Medical Society Directory contains their addresses.

BOOK REVIEWS

The Physiology of Exercise. A Textbook for Students of Physical Education. By JAMES HUFF MCCURDY, A.M., M.D., M.P.E. Editorial Preface by R. Tait McKenzie, M.D., M.P.E., LL.D. 242 pages. Price, $3.

The author states that the object of this monograph is to present a practical guide in the physiology of exercise, for students of physical education.

McKenzie, in his Editor's Preface, points out that McCurdy, through his long interest and wide practical experience, is the logical writer for a work of this sort, an authoritative book which gives facts as they are now known, a book to which educators may direct the earnest searcher after truth.

McCurdy rightly remarks that the person who fails to get during youth a bodily education is handicapped throughout later life. Equally correctly he observes, in regard to exercise, that if given free choice without any instruction most people do nothing.

Technically the book is well arranged for teaching purposes. Part I, on the General Effects of Exercise upon Bodily Function, occupies the first two-thirds of the book. The third portion of the book, Part II, is devoted to a presentation of data on the Effects of Special Types of Exercises upon Bodily Functions. Each individual chapter is a unit, supplied with its own bibliography and questionnaire. The volume itself is well indexed.

The first chapter in the book, entitled "Physiology of Exercise as an Integral Part of Physical Education," deserves wide reading by those concerned with the physical welfare of the communities in which they live. McCurdy concludes as follows:

"Physical exercise is related to physical efficiency. The present civilization is making great demands npon the vitality of the race. School practices whicn train simply eye, ear, tongue and hand do not promote the health of the pupils. Laboratory work, shop work, military drill and domestic duties increase only slightly the big muscle activity. The active use of the big muscles is essential to the health of individuals. These activities are not secured in the home, on the street, or in ordinary business. Big muscle activities are essential to the development of vocational and other kinds of skill. The higher levels of the nervous system depend for their health and stability upon the organic development of the middle and lower levels of the nervous system. Big muscle activity in physical recreation is an essential part of emotional control in relation to the formation of character."

Lectures on Pathology. By LUDWIG ASCHOFF. 365 pages. New York: Paul B. Hoeber. Price, $5.

Aschoff is Professor of Pathologic Anatomy at the University of Freiburg, Germany.

The fourteen lectures contained in the book were delivered in the United States in the spring of 1924, and include the Edward G. Janeway lectures of the Mt. Sinai Hospital, New York; the Lane Lectures of the Leland Stanford Medical School, San Francisco; the Osler Memorial Lecture of the County Medical Association in Los Angeles, and the Harvey Lectures, New York. Other lectures included were delivered before various medical bodies throughout the country.

I. Reticulo-Endothelial System (Janeway Lecture, New York).
II. The Pathogenesis of Human Pulmonary Consumption (Janeway Lecture, New York).
III. Concept of Inflammation.
IV. Pathological Fatty Changes (Lane Lecture, San Francisco).
V. The Normal and Pathological Morphology of the Suprarenals (Lane Lecture, San Francisco).
VI. Atherosclerosis (Lane Lecture, San Francisco).
VII. Ovulation and Menstruation (Lane Lecture, San Francisco).
VIII. The Orthology and Pathology of the Extrahepatic Bile Passages.
IX. The Origin of Gall Stones.
X. The Site of Formation of Bile Pigment (Lane Lecture, San Francisco).
XI. Thrombosis.
XII. The Relation of Mucosal Erosions to the Development of Ulcer of the Stomach (Osler Lecture, Los Angeles).
XIII. The Goiter Problem, Especially the Goiter of Puberty. A Morphological Study.
XIV. Renal Secretion and Renal Diseases (Harvey Lecture, New York).

These lectures are written in a flowing style and are adapted to the understanding of the ordinarily well read medical student or physician. Aschoff has considered each of his subjects from all standpoints, the physiological, chemical, physical and morphological, and the lectures admirably present the present-day knowledge on the various subjects. In the opinion of the reviewer it is a real service to present such a volume to the medical public, and it is one of the few volumes that the reviewer has read with genuine pleasure and sustained interest. Writers of textbooks would do well to study Aschoff's presentation and particularly his success in correlating knowledge from different sources.

While laboratory workers in physiology and pathology may not agree with Aschoff's evaluation of some of the work he quotes in drawing conclusions, the book for them should prove stimulating and a valuable one to pass on to students in their subjects. The average physician and surgeon will do well to peruse the lectures carefully, not only for the pleasure and information but for the grasp to be obtained of the trend of present-day investigative work in pathology and related sciences